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HIDDEN PARATHYROID ADENOMA

Report of a Case

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DURING the past quarter century continued interest in functioning parathyroid tumors has been manifested and the clinical entity is now well established. There is general agreement that desirable results follow adequate surgical removal of the hyperfunctioning tissue. Improved knowledge of altered calcium and phosphorous metabolism resulting from such tumors, their frequent association with urinary tract calculi, and better awareness of the clinical syndrome have led to a higher number of suspected and diagnosed cases by both internists and surgeons. The surgeon, however, may encounter considerable difficulty in locating a parathyroid adenoma in certain patients who have clinical and biochemical evidence which substantiates the diagnosis.

Two such cases are reported by Spingarn and Geist[7] in which the diagnosis seemed beyond question but in which no tumor was found. Cope[3] cites 9 postoperative cases referred to Massachusetts General Hospital for re-exploration after failure to find an adenoma. In each of the 9 a large tumor was found, 8 at the time of subsequent surgery and 1 at autopsy, all presenting surgical problems by virtue of their unusual anatomical locations. Some patients have had two[3] or three[2] explorations before the adenoma was located. Cases have been reported with the adenoma found anterior to the thyroid, along the carotid sheath, in the anterior and posterior mediastinum and at various ectopic sites in the neck, all of which might be expected in view of the embryonal development of the

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parathyroids and thymus from the third and fourth branchial clefts with subsequent descent of the thymus into the mediastinum.

The purpose of this report is to point out another unusual location for parathyroid adenoma, *viz.*, within the substance of the thyroid gland, and to add 1 case to the previously reported small number in this location. Extensive anatomic, pathologic, and embryologic studies have been reported as showing that parathyroid tissue does not truly occur within the thyroid parenchyma. Some workers[1] who have found parathyroid tissue within the thyroid gland point out the presence covering the parathyroid gland of a thin layer of fascia continuous with, and apparently derived from, the thyroid capsule. This supports the contention that the parathyroid became imbedded in the thyroid by a process of invagination. The disagreement in the literature regarding this point seems of only academic interest, in view of the fact that both normal and adenomatous parathyroid tissue has been definitely found in the thyroid gland, obscured from gross observation. This is reported by several workers, Lahey and Cattell,[4] Wellbrock[8] Norris.[6] True parathyroid adenoma, found completely surrounded by thyroid tissue with the necessity of either incision or partial excision of thyroid parenchyma to demonstrate the tumor, occurs uncommonly, but it is of sufficient frequency to warrant awareness of the possibility on the part of the surgeon who makes an exploratory cervical and/or mediastinal search for such a hyperfunctioning tumor. Nine such adenomas, "hidden" within the substance of the thyroid, have been collected by Norris[6] and two subsequently reported by Black and Haynes.[1] The following additional case report is submitted:

W. H. V., a 55 year old white male farmer, was admitted to the Veterans Administration Hospital, Chamblee, Ga., on Aug. 24, 1951, as a transfer from a private hospital with a diagnosis of parathyroid adenoma. The admission history centered about complaints of extreme fatigability, melancholia, and increased irritability of one year's duration, with a seven month history of epigastric burning pain usually noted one and one-half to two hours before meals and relieved by food. The usual normal daily bowel habit had changed to severe constipation for which the patient had given himself enemas every two or three days for three months prior to admission. A weight loss of 10 pounds in the six to eight months preceding admission was claimed. History was negative for known fractures or passage of urinary calculi. (The patient had been known to have pulmonary tuberculosis of the right upper lobe since 1949.) Studies done in the private hospital during the three weeks prior to transfer revealed blood calcium levels of 15.6 and 15.9 mg. per 100 cc., phosphorous 2.5 and 1.0 mg. per 100 cc., total protein 6.5 Gms. per 100 cc. with albumin/globulin ratio 1.32. The alkaline phosphatase was 83 K. and A. units. The serum potassium was 3.2 mEq./L. on two occasions while sodium and chloride determinations were within normal limits. After a measured daily intake of less than 100 mg. of calcium for four consecutive days, the patient excreted more than 500 mg. of calcium in a 24 hour urine specimen

on the fifth day. Roentgenologic examination of the upper and lower gastrointestinal tract, a gallbladder series, and retrograde pyelograms revealed multiple, bilateral, small renal calculi, some pyelonephritis bilaterally, and an area of irritability and spasm in the mid trensverse colon compatible with tuberculous colitis.

After transfer to this hospital, the laboratory values were essentially the same with calcium levels of 15.0 and 13.2 mg. per 100 cc., phosphorus 2.2 mg. per 100 cc. Postero-anterior and lateral chest roentgenograms revealed the known tuberculous lesion in the right upper lobe but failed to demonstrate a mediastinal mass.

The patient was prepared for surgery and operated upon on Sept. 7, 1951, via a low transverse cervical incision. Thorough systematic exploration was carried out from below upward, beginning on the right side. Adequate exposure of both poles was obtained bilaterally and the middle thyroid vessels sufficiently freed to permit rotation of the lateral lobes medialward. Two definite parathyroid glands of normal appearance were noted and a biopsy specimen was taken from one for confirmation. The lower portion of the left lateral thyroid lobe appeared asymmetrical, being slightly larger, and was cystic to palpation but the possibility of an intrathyroid parathyroid adenoma was not seriously considered at this time. The dissection was carried downward along the carotid sheaths into the superior mediastinum. A longitudinal midline extension of the transverse cervical incision was made, using the sternal splitting technic. This incision was carried down to the level of the fourth interspace. Through the combined incision careful anterior mediastinal exploration was performed. The thymic remnants were removed but were found to contain nothing resembling an adenoma. Two small venous channels were traced from the neck into the mediastinum where they terminated in the innominate vein. Nothing resembling an adenoma was seen.

Before closure of the incisions, the previously noted cystic left lower lobe of the thyroid was incised and found to contain clear fluid. The left inferior thyroid vessels were then ligated and the lower portion of the left lobe removed. Histologic study of this specimen revealed the presence of a rather large clear cell parathyroid adenoma within this lobe of thyroid. The tumor measured 3.0 by 2.0 by 1.0 cm.

On the third postoperative day the serum calcium had dropped to 9 mg. per 100 cc. and the phosphorus was 2.5 mg. per 100 cc. On the fifth postoperative day the calcium was 8.7 mg. per 100 cc., the phosphorus 3.4 mg. per 100 cc. On the ninth postoperative day the calcium was 9.3 mg. per 100 cc., with the phosphorus 4.4 mg. per 100 cc. The patient was discharged afebrile and ambulatory on the tenth postoperative day.

Summary

Difficulty in locating some parathyroid adenomas due to their wide variability in location has been reported by several workers in the past.

Other cases are recorded in which the tumor was not located until the second or third exploratory operation had been performed.

Various sites in the upper and lower neck and both the anterior and posterior mediastinum have been sites of those adenomas.

The practical surgical importance of realizing that a parathyroid adenoma may be completely concealed by surrounding thyroid tissue is emphasized.

A case of functioning intrathyroid parathyroid adenoma successfully treated by partial thyroidectomy is reported. It is believed that this is the twelfth such case to be reported in the literature.

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CHOLEDOCHODUODENAL FISTULA

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CHOLEDOCHODUODENAL fistula is one of the spontaneous internal biliary fistulas infrequently encountered in surgery of the biliary and intestinal tract. A brief review of the literature revealed no collection of cases. This type was reported with all internal biliary fistulas. Only an occasional case was found. The first case of spontaneous internal biliary fistula diagnosed by roentgenologic examination was reported by Hunt and Herbst in 1915 after which many more followed. Garland and Brown[6] reviewed 90 cases proved by roentgenologic examination through 1942. In their group there were 24 cases of choledochoduodenal fistula, 19 of which were due to duodenal ulcer. Kehr encountered 100 internal biliary fistulas during the performance of 2,000 cholecystectomies and Puestow[10] found that 3.5 per cent of patients having operations on the biliary tract had this complication. Judd,[9] in reviewing 153 cases of spontaneous internal biliary fistula at Mayo Clinic found only 1 case of fistula connecting the common duct and duodenum.

INCIDENCE

Various authors report the incidence of choledochoduodenal fistula to vary from 6 per cent to 33 per cent of all spontaneous internal biliary fistulas. The largest collection of cases proved surgically was reported by Waggoner[12] and included 819 with the following incidence table:

Cholecystoduodenal	51%
Cholecystocolic	21%
Choledochoduodenal	19%
All others, cholecystogastric and others	9%

PATHOLOGY

The pathologic etiology of internal biliary fistula is amply outlined by Weinberger[13] in order of frequency:

1. Penetration of gallstones from gallbladder or common duct into duodenum or other part of hollow viscera.
2. Extension of an inflammatory process by continuity from the gallbladder or common duct to the duodenum or vice versa, for example, duodenal ulcer.

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3. Primary lesion of the colon, for example, malignancy.
4. Operative, for example, cholecystoduodenostomy.

Symptoms

No typical set of symptoms or signs is present with internal biliary fistula. Those present usually mimic the parent pathology from which they arise. Puestow[10] has stated that symptoms of the parent lesions have subsided following the formation of a fistula but usually they are intensified, if not at once, later in the course of the disease. His conclusions were based on the 3.5 per cent found in 2,000 cholecystectomies.

Diagnosis

The correct preoperative diagnosis of internal biliary fistula can only be made by roentgenologic examination. Borman and Rigler[3] reviewed 267 cases and found that only 86 were recognized preoperatively and in each case the diagnosis was made by the roentgenologist. The presence of gas or ingested barium, or both, in the biliary radicals is presumptive evidence of internal biliary fistula. If there is no history of operative formation of a fistula and no regurgitation through a relaxed sphincter of Oddi, a definite diagnosis can be made. The relaxed sphincter can be ruled out by fluoroscopic observation or by serial roentgenographic films where there is no retention of barium in the biliary tract. This is in striking contrast to the choledochol retention seen in all cases of fistula. The presence of a normally functioning gallbladder without stones, and the presence of a fistula in that area suggests it to be a choledochoduodenal fistula, probably of peptic ulcer origin.

The following case report is presented which illustrates the various features present noted above and adds another authentic case to the literature.

Case Report

K. W., a 28 year old white male, was first seen on Dec. 12, 1949, at Central State Hospital, Lakeland, Ky. Since 1947 he had complained of epigastric pain following meals, associated with frequent vomiting. On two occasions there was severe upper gastrointestinal tract hemorrhage to the point of shock. The last hemorrhage occurred in November, 1949. He had never had jaundice.

Physical Examination: His weight was 125 pounds, blood pressure 100/38 and pulse 82 per minute. The patient was malnourished, pale, and in poor general condition. His abdomen was soft and flat, but there was tenderness to palpation in the right upper quadrant. The remainder of the physical examination was not remarkable.

Roentgenologic Examination: Upper gastrointestinal series: The chest was normal on fluoroscopic observation. The esophagus was normal in course and outline. The stomach was greatly enlarged and the rugal folds were moderately hypertrophied. There was temporary spasticity of the pylorus but barium was manipulated into the duodenum. Shortly after entering the duodenum, a small trickle of barium from the main column was observed to pass through an accessory pocket and into the common bile duct and biliary radicals. There was constant spasticity and irritability of the duodenal bulb and the first portion of the duodenum. The patient was extremely tender over the duodenal bulb. A roentgenogram made after a one and one-half hour interval showed a small amount of barium remaining in the stomach and in the accessory pocket. The remainder of the barium was scattered in the small intestine (fig. 1).

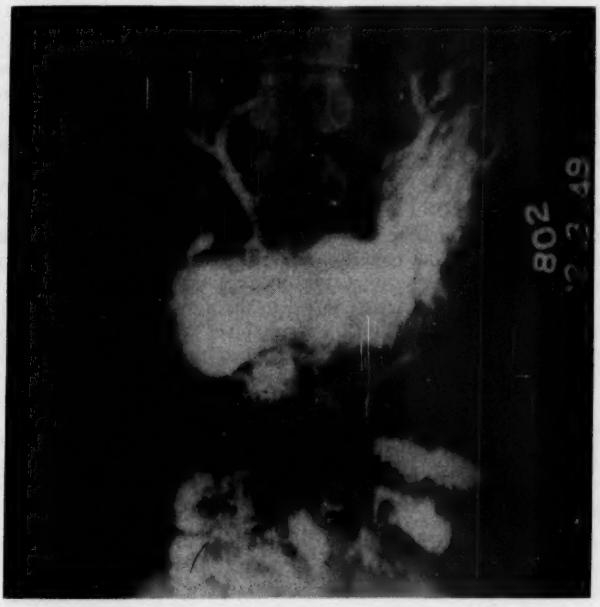

Fig. 1. Enlarged stomach with hypertrophied rugae. Duodenal bulb spastic and not well filled. Fistulous tract between duodenum demonstrated showing filling of common duct, cystic duct, right and left hepatic ducts.

The impression of the roentgenologist was a duodenal ulcer with accessory pocket and rupture forming a fistulous tract with the common bile duct. The barium enema was negative.

Laboratory examinations on Dec. 1, 1949, were as follows: polymorphonuclear leukocytes 6,600 per cu. mm., erythrocytes 2,570,000 per cu. mm., hemoglobin 6.5 Gm. per 100 cc. Urinalysis—color, cloudy yellow, specific gravity, 1,020, reaction 6.0 degrees, sugar negative, albumin negative, acetone negative, bile negative. Microscopic—2-4 pus cells per high power field. Gastric analysis—free hydrochloric acid 21.5 degrees, total acidity 39.3 degrees, free HCL 30.2 degrees, combined HCL 9.1 degrees, acid salts 8.7 de-

grees, occult blood 2 plus. On December 20 the laboratory findings were: erythrocytes 4,470,000 per cu. mm., hemoglobin 10.5 Gm. per 100 cc., total protein 6.8 mg. per 100 cc., specific gravity of blood 1.051, specific gravity of plasma, 1.027.

The preoperative preparation included repeated transfusions, a high caloric and vitamin regimen and continuous gastric suction for several days.

On Dec. 21, 1949, a partial gastrectomy and partial duodenectomy with anterior gastroenterostomy and cholecystoduodenostomy was done. The operative procedure was as follows: under spinal anesthesia the abdomen was opened through an upper transverse incision. The mesenteric attachment of the transverse colon at the hepatic flexure was incised near the reflection. The upper right colon was retracted inferiorly, exposing the first and second parts of the duodenum. The first and second portions of the duodenum and the head of the pancreas were then mobilized. The gastrocolic ligament was then divided from the duodenum to the upper third of the stomach. The right and left gastroepiploic vessels were divided and ligated. The gastrohepatic ligament was incised and the right and left gastric vessels were divided and ligated. The posterior portion of the first part of the duodenum was dissected free from the pancreas and the fistula was located and found to communicate between the common duct and the posterior part of the duodenum. A penetrating ulcer was found on the posterior wall of the proximal duodenum. The fistula was then opened. The duodenum was divided distal to the ulcer and fistula, at the junctions of the first and second portions of the duodenum. The fistula was traced into the pancreas and the region of the common duct. It was then closed with interrupted cotton sutures near its entrance into the duct. The gallbladder was anastomosed to the duodenum using an inner layer of intestinal chromic catgut and an outer layer of interrupted mattress type silk sutures. The stomach was then divided at the junction of the upper and middle thirds and an antecolic Hoffmeister-Polya gastrojejunostomy was performed. The wound was closed in layers with interrupted cotton sutures. The skin was closed with interrupted silk sutures.

The pathologic specimen showed an ulcer of the first part of the duodenum and chronic inflammatory changes of the lymph nodes. There was no evidence of malignancy.

During the postoperative period the patient developed massive pulmonary atelectasis, requiring aspiration with a tracheal catheter which brought immediate relief. The postoperative course was otherwise uneventful. At no time did he show jaundice or evidence of chalongitis. There were no further hemorrhages, abdominal pain, or other symptoms. He progressively gained weight.

At a follow-up examination on June 3, 1950, the patient weighed 146 pounds. His general condition was good. He was asymptomatic. The laboratory findings were: erythrocytes 5,090,000 per cu. mm., polymorphonuclear leukocytes 9,800 per cu. mm., hemoglobin 90 Gm. per 100 cc. A repeat gastrointestinal series on July 21 showed the esophagus, the remaining portion of the stomach, the gastroenterostomy stoma, and the loop of the duodenum to be normal in appearance. No evidence of a fistulous tract was seen.

Discussion. The surgical management must correct two separate but associated surgical entities, *i.e.,* duodenal ulcer and internal biliary fistula. Primarily a poor surgical risk, the patient must be properly prepared. Dehydration, secondary anemia, and malnutrition require correction. In the case presented, this required approximately three weeks. Repeated transfusions were

given, a high protein and caloric intake was established, and temporary medical management of the duodenal ulcer was carried out. Fortunately there was no liver damage or cholangitis. The repeated hemorrhages indicated surgical intervention. In the choice of a surgical procedure the following were considered:

1. Subtotal gastrectomy, leaving the fistula intact.
2. Subtotal gastrectomy and partial duodenectomy with closure of the fistula.
3. Subtotal gastrectomy, partial duodenectomy with closure of fistula and cholecystoduodenostomy.

The first procedure was considered inadequate because the ulcer could not have been resected and the ulcer and fistula might, or might not, have healed subsequently.

The inflammatory process surrounding such an ulcer would very likely heal with considerable fibrosis and probably stricture of the common duct at the site of the fistula. This consideration led to the rejection of the second procedure and made the third the procedure of choice.

The excision of the fistula at the common duct required dissection through the head of the pancreas. The closure was readily accomplished but at an uncertain level in relation due to common duct and to the marked induration of the pancreas. The duodenal stump appeared to be the ideal site of anastomosis with the gallbladder. Since a Hofmeister Polya type of gastrojejunal anastomosis was done, the likelihood of reflux of intestinal contents into the biliary system was diminished.

Conclusion

A case of choledochoduodenal fistula complicating a penetrating duodenal ulcer and its management has been discussed.

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POTASSIUM DEFICITS IN SURGICAL PATIENTS*

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AN understanding of water and sodium chloride requirements and therapy has become widespread in recent years among the practitioners of surgery. Only recently, however, has it been realized that potassium also must be given routine consideration in the planning of fluid and electrolyte management in major abdominal surgery. The addition of a flame photometer to our surgical laboratories early in 1950 permitted us for the first time to obtain accurate, rapid, and repeated studies of the plasma electrolytes in a large number of patients. This has been followed by an experience that has increased our awareness of potassium deficiency, its serious consequences if unrecognized or untreated, and finally our ability to handle the problem. With this background we now feel that very few surgical cases should develop potassium deficits of sufficient severity to produce marked clinical or electrocardiographic manifestations. Darrow[1,2,3] has been chiefly responsible for bringing to light much of the physiologic and therapeutic data concerning this essential cation. The theoretical basis for potassium administration to surgical patients has been well established by other investigators.[4,5,6] It is our purpose to review this experience on a general surgical service over a 20-month period, stressing the frequency of potassium deficiencies, the mechanisms responsible for this state, etiologic factors, clinical and laboratory manifestations, and finally management.

STATISTICAL DATA

During the 20-month period, January 1950, through August 1951, a total of 4,740 determinations of plasma potassium levels were made on 1,358 patients; and one or more abnormally low values were found in 265 cases, an incidence of 19.4 per cent. The normal range of plasma potassium levels as determined by our laboratory varies between 3.5 and 5.0 milli-equivalents per liter (mEq./L.). Of the 265 patients with low potassium levels, the values ranged between 3.0 and 3.5 mEq./L. in 180. Analysis of

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these cases failed to show any manifestations which could be attributed solely to potassium deficiency. Eighty-five were below 3.0 mEq./L., comprising 6.2 per cent of the total patients studied; and it is in this group that significant clinical and electrocardiographic manifestations became apparent. Seventy-two of these 85 cases with markedly low values were surgical patients, and the remainder of our analysis will deal only with these. In 27 of these 72 cases a potassium deficit was present on admission or preoperatively, while 48 developed this finding postoperatively.

Analysis of Mechanisms

In the 72 surgical cases loss of gastrointestinal fluids accompanied by absent or very meager intake of food have been the most important factors resulting in low potassium levels. This has been true whether the deficiency state was present on admission or preopera-

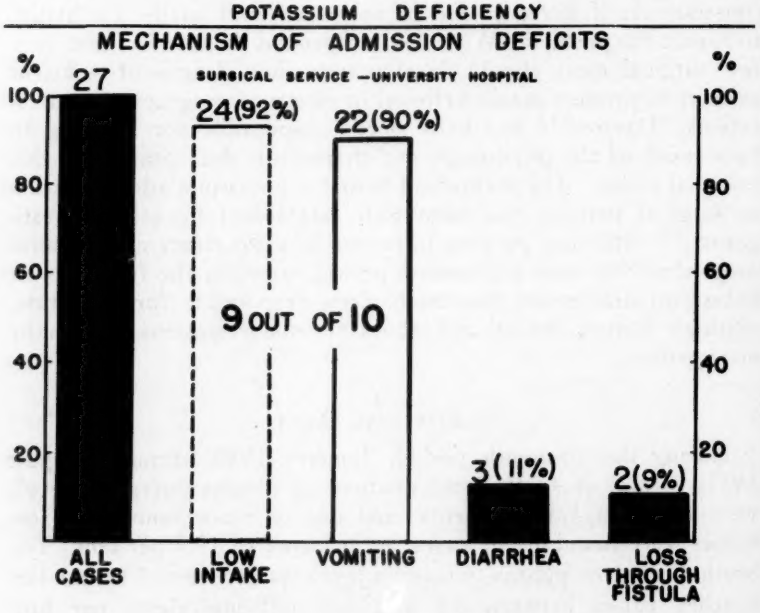

Fig. 1. Mechanism of admission potassium deficit. The major role of loss of gastrointestinal fluids as a cause of potassium deficiency is clearly demonstrated.

tively, or whether it developed following some major surgical procedure. Of these 27 cases in the first mentioned group, 22 had been vomiting for at least several days. The remaining 5 cases were also losing potassium by way of the gastrointestinal tract, 3 with severe

diarrhea and 2 with gastrointestinal fistulas (fig. 1). Deficient intake was an additional factor in 24 of these cases. Obstruction was the responsible etiologic factor in 15 of this group. Pyloric obstruction was present in 9 instances and small bowel obstruction in 6 (fig. 2).

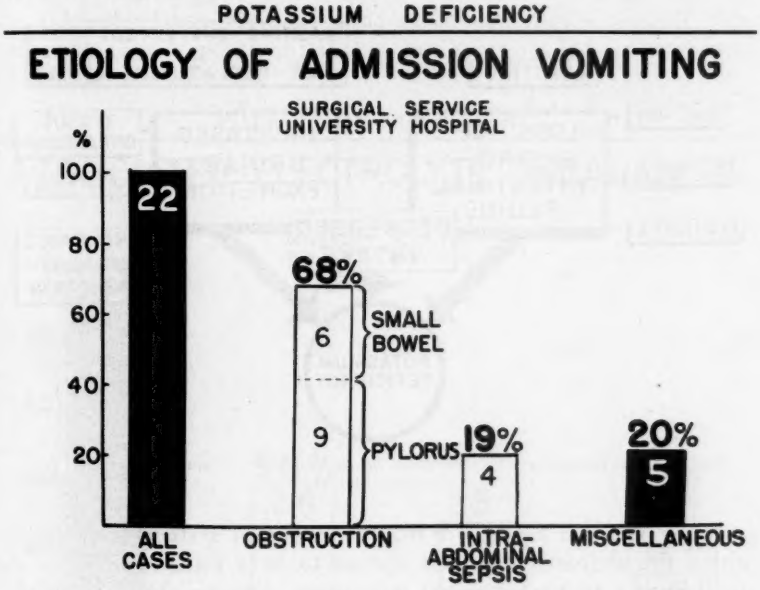

Fig. 2. Etiology of admission vomiting. Obstruction was the responsible factor 15 of the 22 cases.

In the 48 cases manifesting hypopotassemia postoperatively, loss of gastrointestinal fluids by suction with, of course, no oral intake of food, and the administration of parenteral fluids containing little or no potassium were almost invariably forerunners of this situation. Of the 27 patients with low potassium levels on admission or preoperatively, only 3 showed up in the postoperative group, probably indicating that proper therapy, both active and prophylactic, usually resulted once the problem was recognized.

Analyses of urinary potassium in several patients with low plasma levels enabled us to confirm previous observations that potassium continues to be excreted in the urine in appreciable amounts even in a deficiency state. This continued urinary loss is a further mechanism in the production of potassium deficits. We have not evaluated the factor of stress with adrenocortical hyperfunction as a consequence of surgical trauma, producing the so-called "alarm reac-

tion," but others feel that this mechanism with increased loss of potassium in the urine is of major significance[6] (fig. 3).

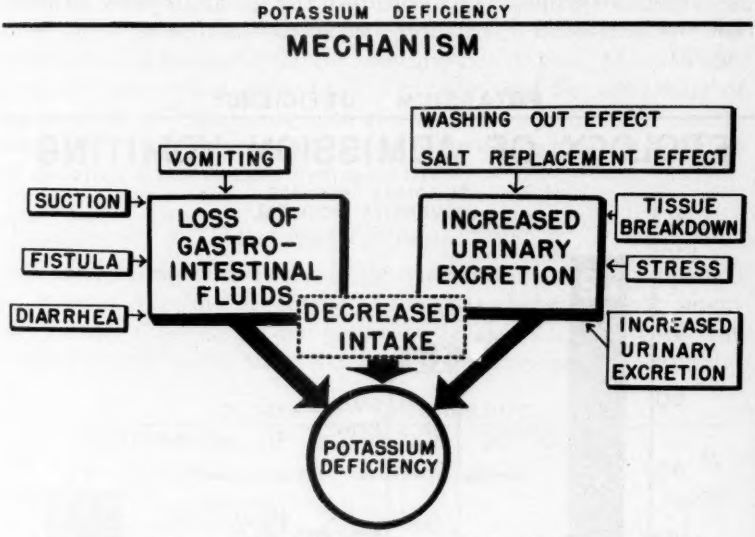

Fig. 3. Diagrammatic representation of the multiple factors responsible for potassium deficits.

It has been our experience that the organ or system operated on within the abdomen does not appear to be a significant factor in predisposing to postoperative potassium deficits. Any major abdominal procedure may be complicated by such a deficiency in the postoperative period, if the various mechanisms noted above come into play.

Manifestations of Deficiency

While the clinical manifestations of marked hypopotassemia may be striking, they are not specific; and a knowledge of the circumstances under which potassium deficiencies occur remains the most useful clinical guide to their detection. Weakness, lassitude, and lethargy are the most frequently noted signs, and these were of sufficient severity to be noted in the charts of 53 of the 72 patients in this series. Abdominal distention was the second most useful sign, being apparent in 36 or 50 per cent of the cases. It is obvious that these signs may be associated with many conditions other than a potassium deficit; and though their frequency may be quite evident in retrospect and from a statistical standpoint, at the time the observations are made their significance is frequently not appreciated. Early in this study there were several instances of a persistent ileus

due to a potassium deficit that were treated with prolonged intestinal intubation before the diagnosis was made and corrective therapy instituted.[7]

In addition to the above signs, a significant decrease in the vital capacity became apparent in a number of instances. We have learned to value this latter observation as an indication of a possible complication in postoperative patients, but again its lack of specificity is obvious.

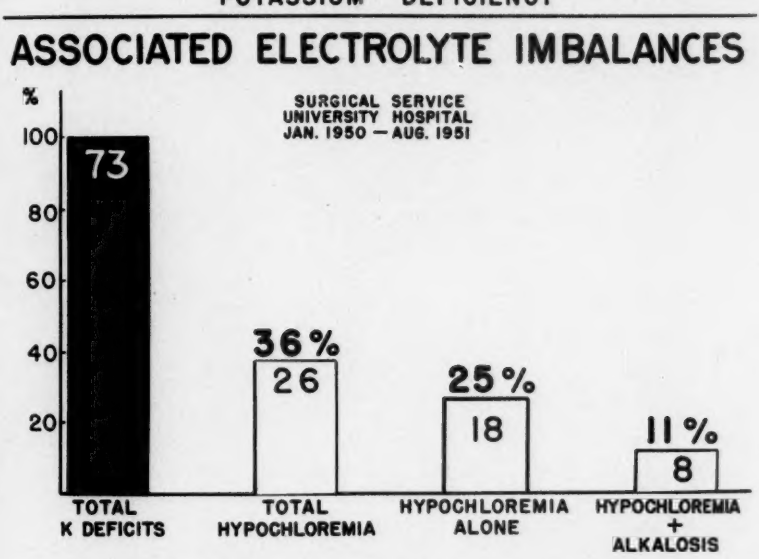

Fig. 4. Hypochloremia with or without alkalosis was an associated finding in 26 of the 72 cases.

Other than the low plasma potassium levels themselves, which we have used as the standard in judging a deficit, the most frequent laboratory finding is hypochloremia. This has been recorded in 26 of the 72 cases, and has been associated with an alkalosis in 8 patients (fig. 4). The persistence of this hypochloremic alkalosis in the face of fluid therapy not incorporating adequate amounts of potassium has been stressed by others. Hence, the presence of hypochloremia and alkalosis in a surgical patient should strongly suggest the existence of a potassium deficit, and indeed these abnormal electrolyte findings will often not be corrected until adequate potassium therapy is added. By and large these laboratory findings do not occur with sufficient frequency to be of much diagnostic aid.

In this study little emphasis has been placed on electrocardiogram for diagnosis, but the typical changes noted by others have been confirmed on several occasions. These changes consist of flattening of the T waves with prolongation of the Q-T intervals.

Therapy

Indications of potassium therapy in surgical patients fall into two principal groups.[8] First, the continued loss of large amounts of gastrointestinal fluids by vomiting, suction, diarrhea or fistula constitutes an important indication for administration of adequate amounts of potassium to replace these losses. Secondly, prophylactic treatment should be carried out in the postoperative period of those patients who have undergone extensive abdominal surgical procedures. This is of particular importance in those patients who must be supported on parenteral fluids or who require prolonged gastrointestinal intubation and suction.

We have used the three preparations first described by Randall, et al.,[4] and have found them quite satisfactory. Their simplicity of preparation and low cost are added attributes. Whenever possible, potassium therapy should be given by way of the gastrointestinal tract, as administration by this route virtually eliminates the danger of potassium toxicity. For this purpose we use an oral preparation containing 3 Gm. of potassium salts, 1 Gm. each of the citrate, acetate, and carbonate, dissolved in 8 cc. of water. This medication is usually given with a flavored beverage. It may also be given by tube feeding or by jejunostomy in appropriate cases. Ordinarily 9 to 12 Gm. of this oral preparation are given in divided doses, although we have used up to 18 Gm. a day in markedly deficient cases with no untoward effects.

Very often, as is apparent from the mechanisms responsible for potassium deficiencies in this group of cases, the oral method of administration cannot be used; and the therapy must be given intravenously. In most instances 30 mEq. of potassium constitute an adequate maintenance dose. This is provided by 2.23 Gm. of potassium chloride; and this dose is easily prepared in 2 ounce bottles by the hospital pharmacy, sterilized by autoclaving, and stored on the wards for immediate use. When the therapy is indicated, the contents of a bottle are dissolved in 20 cc. of distilled water and added to a liter of appropriate fluid, whether this be 5 per cent or 10 per cent dextrose in water or normal saline.

A third preparation, also for intravenous use, contains 6.62 Gm. of sodium chloride in addition to the 2.23 Gm. of potassium chloride. This, too, is placed in small bottles which are sterilized

by autoclaving and stored on the wards. This preparation is useful when replacement of sodium as well as potassium and chloride is indicated.

Ordinarily, as indicated above, 30 mEq. of potassium per day afford an adequate maintenance dose. In those patients with continued loss of gastrointestinal secretions, two or three times this amount is needed. Thirty mEq./L. is a safe and satisfactory concentration and is the one we ordinarily employ; however, occasionally we have used 60 mEq./L. of potassium and intravenous fluid in markedly depleted patients. Even higher concentrations have been used by others with no apparent ill effects.[9]

The chief precaution in potassium therapy is to avoid producing a dangerously high plasma level. Using the preparations and dosage described above, this situation may be encountered only in instances of severely impaired renal function or in marked dehydration. Hence in a depleted patient it is important to hydrate the patient properly with appropriate fluids before adding potassium, and to do this we ordinarily administer 2000 cc. of fluid before giving any potassium. If the urinary output is then adequate, potassium chloride is added to the third liter. Postoperatively potassium is withheld 24 to 48 hours in order that normal renal function may be reestablished; but after that time adequate maintenance amounts are administered daily as long as parenteral fluids or gastrointestinal suction are continued.

Summary and Conclusions

1. Sufficient data has accumulated to indicate the value of potassium therapy preoperatively and postoperatively in many surgical cases.

2. In this series the principal mechanisms responsible for the deficiency state have been loss of gastrointestinal fluids by vomiting or suction and the absence of oral intake. Excluding postoperative factors, the single most prominent etiologic basis has been intestinal obstruction.

3. The clinical and laboratory manifestations of potassium deficits have been discussed. Awareness of situations which may lead to such deficits is the most useful clinical guide to their prevention or detection.

4. The treatment of established deficiencies and prophylactic therapy are outlined, with consideration given to both the oral and intravenous routes of medication. Thirty mEq./L. of potassium (2.23 Gm. of potassium chloride) constitute an adequate daily maintenance dose in most instances. In the dehydrated patient this

amount may be added to the third liter of fluid. At least this amount, and frequently more, should be given daily starting 24 or 48 hours after surgery in those cases who must be supported by parenteral fluids for more than three days, or in whom gastrointestinal suction is used.

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SACCIFORM ANEURYSM OF THE RENAL ARTERY

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ANEURYSM of the renal artery is a rare lesion. Mallory Institute of the Boston City Hospital reported only 1 case in 13,525 autopsies. Kment says that 5 were found in a series of 41,000 post-mortem examinations. In the literature there are 85 case reports that may be accepted as types of aneurysm of the renal artery. In some of these reports the descriptions are not complete enough to permit accurate classification.

Aneurysms of the renal artery have been classified into true and false types. The wall of the true type is formed by some or all of the arterial coats. In the false aneurysm, of which 20 cases have been reported,[67,63,15,54,64,57,30,23,38,53,47,61,37,59,33,80,66,52,7,42] there is rupture of the artery and the extravasated blood becomes surrounded by fibrous tissue which forms the wall of the so-called false aneurysm.

Thus, we find that a false aneurysm is actually a hematoma which is walled off and is certainly not a vascular structure. When cases of true and false aneurysms are analyzed we find that the etiology, symptoms, treatment, and prognosis, as well as pathology of each condition, are quite different.

True aneurysms may be intrarenal or extrarenal. Thirteen cases[20,2,56,33,22,68,71,36,50,25,34] of the intrarenal type have been reported. Some are very clearly shown to be true aneurysms of interlobular arteries which ruptured to produce hematomas.[2,22] Several cases followed renal surgery.[33,34] Rupture of the intrarenal type may result in severe hematuria by communicating with the renal pelvis.

There are several types of extrarenal aneurysm. In 1941 Smith reported a case in which the renal artery was about four times the usual diameter. This may be considered to be a diffuse aneurysm which resulted from a shrapnel wound 16 years before.

Two cases of extrarenal aneurysm are described as being fusiform. Skillern, in 1906, reported a case which was associated with a false aneurysm; a nephrectomy was done. In 1947, Puigvert and Cols described a case in which the opposite kidney was absent and the aneurysm was successfully resected.

Most of the true aneurysms are sacciform. Forty-nine[43,13,28,79,57,21,3,1,18,16,78,38,12,70,76,75,8,11,24,14,82,40,9,81,29,49,83,17,35,51,60,74,77,58,32,31,10,42,46,41,44,25,6,26,5,4,48] case reports are found in the literature and to these 1 new case is added. The first sacciform aneurysm was reported in France by Leudet in 1852. This was a 62 year old woman who died of nephritis. She had been ill for two years and had increasing dyspnea

and edema for two months before death. At postmortem examination, an aneurysm the size of a bean was found at the bifurcation of the right renal artery; it was filled with a semisoft, yellowish material but no blood, and the walls were calcareous. The kidney was less than one-half the size of the left.

Söderlund diagnosed the first case of sacciform aneurysm in 1925 after studying the roentgenograms made on Key and Akerlund's case in 1917 at the same hospital, the Maria Krankenhaus in Stockholm. This was a 63 year old woman who for three months had had dysuria and suprapubic and left renal pain. She had generalized arteriosclerosis. There was no abdominal tenderness and no mass was palpable. There were many pus cells but no red cells in the urine. On roentgenologic examination, in the hilus of the left kidney a nickel-sized, round, sharply defined calcified shadow with less dense center and round, thick border was observed. A diagnosis of calcified left renal aneurysm was made. An abdominal operation was done, hoping to extirpate the aneurysm but nephrectomy was necessary. In the kidney hilus there was a stone-hard, round mass on the superior branch of the renal artery; it had a short pedicle and measured 12 by 15 mm. Distal to it were two branches, one to the anterior and one to the posterior part of the kidney. On the anterior branch was a pea-sized aneurysm which was not calcified. Hemorrhagic infarcts were found in the kidney.

The following case is the twelfth reported sacciform aneurysm of the renal artery to be diagnosed preoperatively.

Case Report

This 41 year old white woman was admitted to the hospital May 24, 1947, complaining of pain in the right costovertebral area and in the right upper quadrant of the abdomen. About seven days before, she rather suddenly began having pain in the lower right chest, posteriorly; it was a constant aching but much worse on breathing. Four days later the pain was very severe and she could hardly breathe. The chest was strapped by her family doctor who thought she probably had pleurisy, but this gave her only slight relief and she was admitted to the hospital three days later. There was no history of previous attacks of such pain or discomfort. She had no digestive symptoms. There was no dysuria and no hematuria.

Four years previously she had pneumonia which was followed by pleurisy. She had never had any accidents or injuries of importance. Twenty years before admission she had a right oophorectomy, bilateral salpingectomy and appendectomy. Three years before, the uterus and remaining ovary were removed; pathological diagnosis was serous cystadenoma of ovary and fibrous perimetritis of uterus. She had one full term pregnancy, resulting in a normal delivery in 1925. The family history was essentially negative.

Examination: This patient, of average size, was not acutely ill. Temperature, 99 F; pulse, 85; respiration, 22. Blood pressure was 118/75. There

was some limitation of expansion of the right chest with impairment of breath sounds of lower half of right lung. There was no enlargement of the heart; rhythm was regular and there were no murmurs. There was marked rigidity in the upper right quadrant of the abdomen and of the right flank muscles with considerable tenderness. There was a suggestive mass in the region of the gallbladder.

The blood count was within normal limits. A catheterized urine specimen was negative. A blood Kahn was negative.

Report of the roentgenologic examination stated: "Preliminary films show two large dense shadows in the right upper quadrant in the region of the kidney and gallbladder. One shows a faint ring-like shadow suggestive of a gallstone and does not shift with the kidney. The other shadow is dense throughout with thickened calcified wall and its contour is irregular in shape. It lies in the soft tissues between the kidney pelvis and the right transverse process of the second lumbar vertebra.

"Intravenous pyelogram: Films show no filling in five minutes. The kidney pelves and calyces are well visualized in 15 minutes. The pelvis of the right kidney is deformed by the dense shadow so that retroperitoneal calcification deformity of the right kidney pelvis is inferred" (fig. 1).

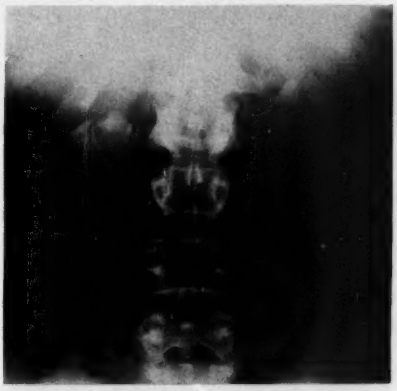

Fig. 1. Intravenous pyelogram showing almost characteristic x-ray shadow of a calcified aneurysm of the renal artery. It demonstrates the extrinsic pressure deformity of the renal pelvis. A gallstone is present overlying the kidney parenchyma.

Preoperative clinical diagnosis: (1) Calcified aneurysm of right renal artery. (2) cholecystitis with lithiasis.

Operation: (1) Nephrectomy, right. (2) Aneurysmectomy, right renal artery. (3) Cholecystostomy.

Spinal anesthesia and intravenous pentothal sodium were given. Upper right paramedian incision was made. After opening the abdominal cavity, the colon was displaced medially, the peritoneum was incised and the right kidney exposed. At the hilus of the kidney a hard mass, approximately 2 cm. in diameter, was found. After it was dissected out, a small area was observed to pulsate. The mass was found to originate from the lower border of the renal artery.

The kidney was normal in size and appearance. The renal vein was doubly ligated and cut; the renal artery was then doubly ligated and severed proximal to the aneurysm. The ureter was cut about 2 inches from the renal pelvis and the kidney with the aneurysm was removed. There was very little bleeding.

Fig. 2. Specimen injected with Diodrast showing the aneurysm and circulatory system of the kidney as well as excretory system.

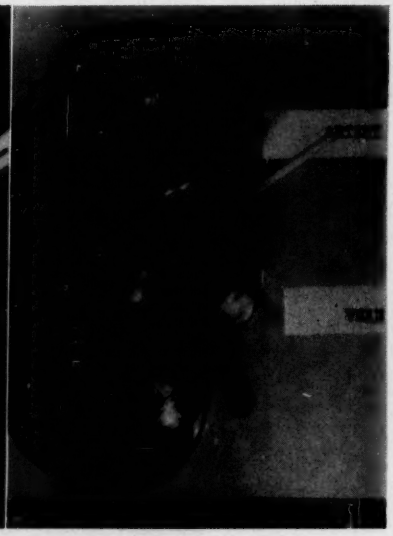

Fig. 3. Specimen showing the aneurysm and markers in the artery and vein.

Fig. 4. The shell-like aneurysm, opened.

The gallbladder was thick-walled, greatly distended with somewhat purulent appearing fluid and contained two large round stones, which were removed. A tube drain was sutured into the gallbladder and brought out through a stab wound to the right of the incision.

The patient had an uneventful convalescence. She was able to void the first day and urine output was quite good. There was no gaseous distention. She was ambulatory from the third day on. The gallbladder tube was removed on the eleventh day. There was practically no drainage from the stab wound on the twelfth day and she was discharged in good condition. She was last seen 20 months later and at that time had no complaints.

Pathology: "Gross Specimen consists of a small kidney which measures 98 by 40 by 30 mm. The renal artery and ureter are attached. There is an aneurysmal dilatation of the renal artery 1 cm. from the pelvis which measured 25 by 20 mm. (figs. 2, 3). The wall of the aneurysm is calcified and forms a thin bone-like shell (fig. 4). On opening the kidney, the paranchyma is very well differentiated and the pelvis is approximately normal in size and possesses a clean, glistening epithelium. Microscopic: Sections of blocks through the parenchyma of the kidney show no remarkable histologic changes. Diagnosis: Calcified aneurysm of the renal artery."

An additional study of the specimen was made. The renal artery divides into two branches, one supplying the superior pole and the other the middle portion of the kidney. The aneurysm arises at the bifurcation without a pedicle so that both branches actually lead off from its wall, making resection of the aneurysm impossible. Sections of the aneurysm and distal branches for microscopic study show subintimal calcification, degeneration of the media with fatty changes and very mild inflammation of the adventitia. Interestingly, at one point there is complete interruption of the media with exvagination of the intima, probably due to rupture caused by enlargement of the aneurysm.

In order to arrive at a clinical picture, the possible etiology, and the best treatment of this lesion, the 50 cases of sacciform and, because there is little difference, the two fusiform aneurysms are studied. Keen described a specimen of typical sacciform aneurysm placed in the museum at St. Bartholomew's Hospital, London, in 1882, but no clinical information is available and so it is not included here.

Twenty-six of the reports are in American literature, nine in German, eight in British, three in Italian and two each in French, in Scandinavian and in Spanish.

Two of the cases are in Negroes[10,4] and the others are in the white race.

Aneurysm of the renal artery is fairly well distributed in all ages. It has been found in two children less than 10 years of age, the youngest five years old. The oldest patient was 82 years. Twenty-nine of the patients (58 per cent) are past 45 years of age, but only 12 are past 60 years. The average age is 47.5 years.

The lesion is more predominant in females. Thirty (60 per cent) were in women and, interestingly, of these, four were pregnant: this is 13 per cent of the women, and 8 per cent of the total patients.

Pathology. This lesion is almost equally common on each renal

artery, there being 24 on the right and 25 on the left. In Healey's and Carson's cases the aneurysms were bilateral. Healey's case also contained multiple aneurysms on each artery and, in addition, the cases of Söderlund, Lennie and Sheehan, and Brady and Hanten contained multiple aneurysm of one artery. Howard, Forbes and Lipscomb's case is described as being sacculated and multilocular. Smith and Walkling reported a 7 mm. calcified saccular aneurysm of a superior accessory renal artery on the right side. In many instances it is not clearly stated whether the aneurysm is of the main artery or of a branch. In 19 cases (38 per cent) it is reported to be of one of the branches, usually quite near the bifurcation. In 8 additional cases the aneurysm is found at the bifurcation.

Many of the aneurysms are reported to be the size of a pea or a hazel-nut, while one was the size of a child's fist and two the size of an orange. Accurate measurements are given for 23 of the aneurysms and these average 18.6 mm. in greatest diameter. Probably the largest measured specimen was a 70 mm. saccular, calcified aneurysm reported by Solis-Cohen and Steinbach.

Half of the sacciform aneurysms are stated to be calcified. Seventeen of the 25 (68 per cent) were in patients past 50 years of age and only three were in patients under 40 years of age. The youngest patient in whom a calcified aneurysm was found was Barnard's patient, a nine year old boy. The oldest patient with a calcified aneurysm was 76. In 14 cases (28 per cent) arteriosclerotic or atherosclerotic changes in the arteries are mentioned. Periarteritis nodosa is described in 2 cases. In Barnetson's case tuberculosis of the aneurysm was found; it is quite likely that this was secondary to a widespread tuberculosis. Syphilitic pathology is not described in any case.

A study of associated pathology is important. Circulatory changes of the kidney, such as atrophy and infarcts, are described in 9 cases (18 per cent), and in 1 of these there is a very striking narrowing of the lumen of the artery just distal of the ostium of the aneurysm.[25]

Calculi are found in 4 cases. Four patients had pyelonephritis, and of these 2 had carcinoma of the prostate. In 1 case of aneurysm the kidney was of a horseshoe type. There was 1 case with a hypernephroma and another with an adenoma of the kidney. In 13 cases a perineal hematoma or a false aneurysm was described as being associated with the true.

Etiology. Trauma has always been considered to be the most common cause of aneurysm of the renal artery. It has been thought that a blow may damage and weaken the walls of a vessel severe

enough so that there is immediate formation of an aneurysm, or that the damage may be less so that an aneurysm may gradually develop in the months to follow. It is doubtful, however, if trauma would result in the development of this lesion in a normal vessel. It is much more likely that aneurysms develop in vessels that are congenitally defective and the congenital weaknesses are much more likely to be found at or near the bifurcation of a vessel, where many of the aneurysms are found.

If trauma does play a role, then formation of the aneurysm usually is gradual. There are only 2 cases in which there was immediate development of a true aneurysm but it is not possible to say that the aneurysm did not exist before the trauma. In Hilton's case the patient was struck by the buffer of an engine and died on the twenty-sixth day from hemorrhage. There was an intact true aneurysm and also a false aneurysm as well as a peritoneal tear resulting in hemorrhage into the abdominal cavity. Turner's patient fell from a small cart and died on the thirty-first day; he had a perirenal hematoma as well as a small true aneurysm.

In 5 other cases[1,72,51,73,6] there is a history of old injury from two years to 22 years before, and in some of these cases there was also associated chronic nephritis and arteriosclerosis, which could have played a part in the etiology.

Localized and generalized arterial disease are probably very important in explaining the cause of these aneurysms. Unfortunately, however, in only a few cases is there a microscopic study of the arterial structure. Degenerative changes would certainly favor the formation of aneurysm. Fourteen (28 per cent) of these patients are specifically stated to have generalized arteriosclerosis and in many of the others there is local calcification. Hypertension is mentioned in 11 (22 per cent) cases and one of these is of the Goldblatt type,[32] which definitely improved after nephrectomy; others showed some improvement after the aneurysm was removed. Periarteritis nodosa was almost conclusively shown in 2 cases[19,25] to be the etiology. In 1 case[3] it was thought that embolus from stenosis of the mitral valve caused the aneurysm. Syphilis quite definitely does not play a part in the etiology. In 11 cases it was stated that the Wassermann was negative. Onell and Valencia's case probably is the only one that had syphilis, which was congenital, but only at times was the Wassermann positive.

History and Examination. In 10 cases (20 per cent) there were no symptoms from the aneurysm and the lesion was found incidentally at postmortem examination.

Most clinical descriptions of this condition state that the characteristic picture is abdominal pain followed shortly by hematuria and later by a tumor.

Pain is mentioned in 35 cases (70 per cent). It is described as being dull, gnawing, colicky, or even throbbing. Invariably, however, the pain is due to an associated lesion or to a complication, and not to the aneurysm itself. In many cases the pain is unquestionably due to renal lithiasis, kidney infection, or cholecystitis; in other cases the discomfort is probably due to pressure symptoms or secondary circulatory disturbances.

Hematuria, the next important sign mentioned, was found in 17 cases (34 per cent). This was usually found to be due to stones, tumor, infection or trauma. In Shramm's case a stab wound established a communication between the sac of the true aneurysm and the renal pelvis resulting in severe urinary hemorrhage which continued intermittently for over a year. Only in 1 case was no other possible cause for the hematuria given. And so, we might say that hematuria is not a sign of sacciform aneurysm of the renal artery.

A palpable tumor was found in only 2 uncomplicated cases. On examination of Solis-Cohen and Steinbach's patient, an expansible lemon-sized mass transmitting thrill and bruit was found near the lower pole of the ptosed right kidney. This proved to be a 70 mm., saccular, partly calcified aneurysm of the inferior branch of the right artery. In Heriot's case there was a pulsating tumor medial to the left kidney and this was also found to be an aneurysm. On the other hand, there is usually a palpable mass and frequently a picture of shock when there is hemorrhage and a resulting false aneurysm.

Thus, we find that uncomplicated aneurysms usually produce no symptoms and that the triad of symptoms mentioned is usually found in cases with rupture of the aneurysm. These aneurysms are discovered at postmortem examination or after the complication of hemorrhage results.

Diagnosis. True aneurysm is rather difficult to diagnose. Twenty-six of the 52 cases were found at autopsy. Ten of these were found incidental to other causes of death, such as myocardial failure, tuberculosis and carcinoma. In 15 cases hemorrhage from rupture of an unsuspected aneurysm was found to be the cause of death. Three of these were operated upon but the aneurysm was not found until autopsy. In 1 other case[3] there was intra-abdominal hemorrhage from a laceration of the liver, the aneurysm remaining intact.

The roentgenogram picture is the only possible way to diagnose an uncomplicated aneurysm of the renal artery. Key and Akerlund, in 1917, were the first to make a thorough study of the roentgenologic appearance of this lesion. It remained for Söderlund in 1925 to make the first preoperative diagnosis.

An opaque, wreathlike shadow with a more or less transparent central zone found in the renal hilar area is very suggestive. There are many lesions that may give an opaque shadow in this area, but, most important, it must be differentiated from renal stones, calcified lymph nodes and from calcified renal cysts. Levine recommends laminography as an aid in differentiation.

The definite roentgenologic characteristics of a calcified aneurysm are: 1. A round or oval shadow with a more or less transparent center and a dense mulberry-like structure of the periphery, which is the result of irregular calcification. 2. There may be a definite defect in the outline, which is the connection between the artery and the aneurysm. 3. It is located outside the kidney and may cause a pressure deformity of the renal pelvis. Because only half of these aneurysms are calcified it should be pointed out that when there is an extrinsic pressure deformity of the renal pelvis for no apparent cause, the presence of an uncalcified aneurysm of the renal artery should be considered.

Twelve of the 52 cases were diagnosed preoperatively by means of roentgenologic studies. Wesson and Fulmer diagnosed a case but the patient was not treated because of carcinoma of the prostate; the diagnosis was substantiated at autopsy. The 11 other cases were operated on successfully.

Arteriography should be a very important method of diagnosis. R. dos Santos of Lisbon in 1929 reported his method of injecting a 100 per cent solution of sodium iodide into the aorta and this produced excellent arteriograms. At first, this was not considered to be of practical value but since 1942 when Nelson of Seattle reported using an 80 per cent solution, the risk has been considered quite low and the information obtained quite valuable. Diodrast is also an excellent medium for arteriography. To date no cases have been diagnosed by this method.

If a tumor is palpable medial to the kidney, one might be suspicious of an aneurysm, particularly if there is pulsation or bruit. However, this was found in only 2 uncomplicated cases.[77,26]

If there is hemorrhage from the aneurysm there usually will be a clinical picture of shock and later a palpable tumor. In 7 cases there was a history of preceding trauma and in 1 case apparently a

sudden twist of the body seemed to cause the aneurysm to rupture. In 11 cases there was apparently no cause for rupture; 4 of these were in women during late pregnancy. In these cases of rupture it is not possible to say whether the bleeding is from the kidney, renal vessel or an aneurysm. In all cases of rupture of an aneurysm there is a perirenal hematoma, possibly hemorrhage into the peritoneal cavity, or, if communication with the renal pelvis occurs, there is hematuria.

Treatment. It is unfortunate that only the calcified aneurysms can be diagnosed. These are usually not potentially dangerous and it is probably not necessary to urge removal when a calcified aneurysm is diagnosed. However, except for 1 case, surgery has been performed on all patients when the lesion was diagnosed. The average age of this group is 54 years. The average age of cases discovered at autopsy is 64 years. This means a life expectancy of 10 years if nothing is done, and then the patient dies of some other cause. In only 1 case did an aneurysm that was somewhat calcified rupture, and this was at the age of 24 years during late pregnancy.

Surgery is the only method of treatment. One should always have in mind doing a resection of the aneurysm, and so preserve the kidney. Unfortunately, a nephrectomy is usually necessary and this was done in 22 cases.

There was successful resection of the aneurysm in only 3 cases. In 1917 Key and Akerlund attempted to remove the aneurysm in their case, but a nephrectomy was necessary. It remained for Callahan and Schlitz in 1926 to report the first successful resection of a renal artery aneurysm. This was also done by Bonino in 1946. In 1947, Puigvert and Cols resected a fusiform aneurysm in a patient with absence of the other kidney. Smith and Walkling also attempted to excise an aneurysm but because of injury to a branch of the renal vein had to do a nephrectomy.

Great care must be taken in making traction on the kidney during operation, because the aneurysm is medial to the kidney and, at times, quite friable. For this reason it is definitely advantageous to make an abdominal approach. This was specified as having been done in 6 of the cases. In 1937 McKay reported that in his case very little traction caused rupture of the aneurysm resulting in considerable hemorrhage which made a nephrectomy necessary. In Onell and Valencia's case manipulation of the aneurysm caused hemorrhage and the patient died six hours later.

There were 4 cases of exploratory laparotomy in which hemorrhage was found but the aneurysm overlooked. Chisholm drained

the hematoma and this patient died on the second day. Lennie and Sheehan did a cesarean section and this patient died one hour later from hemorrhage from a true aneurysm. Waegner found a hematoma and this patient died shortly after the operation. In Barnard's case a laceration in the liver was packed and the patient died on the second day; the aneurysm was intact.

Two patients on whom a nephrectomy had been done died. Hinman and Olson's patient died in 10 hours from hemorrhage from a suprarenal artery. McKay's patient died on the sixth day from pneumonia and myocardial failure.

Thus, there were 29 patients with aneurysm of the renal artery operated on and there were six[3,11,81,29,51,42] deaths. There were three resections with no mortality. There were 22 nephrectomies with two deaths, a 9 per cent mortality. The other four operations were 100 per cent fatal.

It should be pointed out that the successful surgery was done on patients who had no complications of aneurysm. Of the six deaths, four were in patients with hemorrhage. It should also be mentioned that every case diagnosed as aneurysm preoperatively was successfully operated on.

Prognosis. Twenty per cent of these cases were found incidentally at autopsy and these patients had no symptoms referable to the lesion.

Twenty-four per cent had some discomfort, frequently cystitis, which resulted in diagnosis of the lesion before there were any complications. Eleven of these 12 cases were operated on and the patients survived. The other patient died of carcinoma of the prostate and at autopsy the lesion was proved to be present.

Thirty-two per cent of the cases had complications. The aneurysms may rupture to produce a hematoma or false aneurysm; it may, as a result of injury, rupture and bleed into the peritoneal cavity through a tear; it may rupture into the renal pelvis producing hematuria, which may be severe.

These aneurysms may rupture for no apparent cause. This was true in 11 cases[18,78,76,11,14,29,81,60,58,42] (22 per cent), and it is probably of considerable importance that 4[11,60,58,42] of these were in women who were pregnant and who had had many children; all of these patients died. Engel says that a sudden movement of the body caused rupture of the aneurysm in his 82 year old patient. All of these patients had a hematoma and, in addition, some had hematuria.

Trauma does not seem very likely to cause rupture. This was noted in only 2 cases. Gruber tells of a 39 year old man who fell against a box 10 months before death; there was a large false aneurysm originating from a true aneurysm. Oestreich's patient, a 50 year old woman, who fell downstairs two months before hematuria developed, died one month later of rupture of sacciform aneurysm.

Trauma caused two other peculiar complications. In Barnard's case a 9 year old boy fell from a horse, which caused a calcified aneurysm to lacerate the liver, producing a severe abdominal hemorrhage; packing the liver did not prevent the child from dying. Schramm reported the case of a stab wound in the flank causing communication between an aneurysm and the renal pelvis, resulting in hematuria; the patient was cured by nephrectomy a year after the injury.

Fourteen of these cases with complications died and only 2[72,70] survived because a nephrectomy was performed.

It should be added that these aneurysms can produce secondary circulatory changes in the kidney, such as atrophy and infarcts; these were found in 16 per cent of the cases.

Summary

Aneurysm of the renal artery, which is a rare lesion, has been classified into true and false types. The false type is not a vascular structure and should not be studied with the true type; when cases of each are analyzed together, conclusions are misleading.

Fifty cases of sacciform and 2 cases of fusiform aneurysm are studied. The lesion is fairly well distributed in all ages and is more predominant in women. These aneurysms are usually the size of a hazelnut and half are stated to be calcified. They may cause circulatory changes in the kidney.

If trauma is a cause of aneurysm it is most likely that the vessel was congenitally defective. Localized and generalized arterial disease, such as degenerative changes, are very important in explaining the cause of these aneurysms.

Over 50 per cent of the cases were found at autopsy. The roentgenogram is the only possible way to diagnose an uncomplicated aneurysm of the renal artery. An opaque, wreathlike shadow with a rather transparent central zone found in the renal area is almost diagnostic. Arteriography should be very helpful in diagnosis. If there is hemorrhage from the aneurysm there is usually a clinical picture of shock and a palpable tumor results.

Surgery is the only method of treatment. An abdominal approach is recommended, and a resection of the aneurysm is advisable, if possible.

Twenty per cent of these cases were found incidentally at autopsy. Twenty-four per cent were diagnosed. Thirty-two per cent had complications, usually resulting in a perirenal hematoma; the aneurysm usually ruptures for no apparent cause, and four of these were in women who were pregnant. Only 2 patients survived rupture of the aneurysm and this was because a nephrectomy was performed.

Case Reports

In 1900 Keen and also Morris reported surveys of previous cases. In 1922 Vogeler, in 1923 Conroy, and Gerard in 1930 wrote review articles. Mathé in 1932 tabulated 56 cases, one being simply a museum specimen; he also included the case of Nebel from 1719, which is now considered to be an aortic aneurysm. In 1943 Lowsley and Cannon added 19 more cases. Then, in 1944, Lazarus and Marks reported 1 additional case and tabulated the 75 cases of true and false aneurysms to date. Following are summaries of 10 additional cases of true aneurysm.

Untabulated Cases. Waegner, Alfred (Germany, 1931): A 39 year old male. *History.* Lower abdominal pain and vomiting beginning day before admission. No hematuria or dysuria. No trauma. *Examination.* Abdomen distended. In left upper quadrant dullness down to umbilicus; spasm in left abdomen; severe pain on palpation. Urine: No red blood cells. *Diagnosis.* Probably perforated stomach ulcer. *Treatment.* Upper median incision. Liquid blood in hypogastrium. Mesentery in lower part torn and infiltrated with blood. Hematoma reaches to left kidney. Cause not found. Because of condition, abdomen closed. *Result.* Died shortly thereafter. *Pathology.* Autopsy: Tear in left renal artery. Aneurysm of lower branch of renal artery in hilus with fresh tear. Large subcapsular hematoma and retroperitoneal hematoma.

Lennie, R. A., and Sheehan, H. I. (Scotland, 1942): A 24 year old female. *History.* Severe pain in left loin five hours; collapse; shock. Thirty-six weeks pregnant. Five previous pregnancies. *Examination.* Tender in left flank; dullness. *Diagnosis.* None. *Treatment.* Laparotomy; very large retroperitoneal hematoma. Cesarean section. *Result.* Died one hour later from hemorrhage. *Pathology.* Left renal artery: 2 saccular aneurysms, each about 2 cm. in diameter, one at hilum, other 3 cm. from hilum. Latter one had leaked, resulting in 3 cm. false aneurysm and this ruptured causing hematoma. Atheromatous patches.

Levine, B. (New York, 1945): A 59 year old female. *History.* Pain in right upper quadrant and right loin for 15 years; back pain was constant and boring. Duodenal ulcer three months before. One episode of hematuria two months before admission. *Examination.* Blood pressure: 170/90. Urine: negative. Wassermann: negative. Roentgenogram: wreathlike shadow medial to right kidney. *Diagnosis.* Aneurysm of renal artery. *Treatment.* Nephrectomy, transperitoneal. *Result.* Cured. Hypertension persisted. *Pathology.* 2.7 by 2 by 1.5 cm. calcified aneurysm of renal artery.

Herbut, P. A., and Price, A. H. (Philadelphia, 1945): A 44 year old

male. *History*. Hematuria and dull pain in left costovertebral angle. Six months later had severe headaches and dizziness. Four months later had paralysis and died in coma. *Examination*. When first seen blood pressure ranged from 116/68 to 130/74; urine contained some red cells and pyelogram was negative. Ten months later blood pressure was 170/120; urine contained no red cells, casts present; Wassermann negative; pyelogram negative. Two weeks prior to death blood pressure was 230/110. *Diagnosis*. None. *Treatment*. None. *Pathology*. Necropsy. In main branch to upper portion of left kidney 1 cm. from origin, a 1.5 cm. saccular aneurysm. Lumen of vessel narrowed just beyond ostium to aneurysm. Periarteritis nodosa. Atrophy of upper pole of kidney.

Brady, J. P., and Hanten, J. S. (Mare Island, 1946): A 52 year old male. *History*. Pain in lumbar region for one month. Kicked in left lumbar region 22 years before. *Examination*. Blood pressure 160/100. Roentgenogram: incomplete annular shadow near hilus in the lower pole, left kidney. *Diagnosis*. Calcified cyst; new-growth and renal artery aneurysm considered. *Treatment*. Nephrectomy. *Result*. Recovered. Hypertension not improved. *Pathology*. Two sacculated aneurysms of posterior branch of renal aneurysm. One 2 by 1.5 by 1 cm.; other 2.5 cm. in diameter is calcified. Arteriosclerosis. Pyelonephritis.

Heriot, A. J. (England, 1946): A 49 year old female. *History*. Swelling in left side for five months, increasing in size; now a throbbing sensation. High blood pressure for three years. *Examination*. Kidney palpable and internal to that was pulsating tumor. Roentgenogram suggested that this tumor arose from the renal artery rather than from aorta or splenic vessel. Wassermann: negative. Urine: occasional red cell and granular cast. Blood pressure 235/145 and later 175/95. *Diagnosis*. Aneurysm of renal artery. *Treatment*. Anterior approach. Aneurysm of renal artery freed and short pedicle ligated. Kidney removed with aneurysm. *Result*. Cured. At first blood pressure was normal, but later was again high. *Pathology*. Compression of kidney resulted in extreme ischemia and extensive atrophy; no normal glomeruli seen. Wall of aneurysm consists of edematous fibrous tissue; no endothelial lining.

Bonino, M. (Italy, 1946): A 66 year old female. *History*. Colicky pains in the left flank for 14 years, every three to four months. No urinary symptoms. *Examination*. Urine: Trace of albumin. Roentgenogram: Small circular opacity at the angle formed by the pelvis with the superior calyx of the left kidney. *Treatment*. Aneurysm of artery was ligated and resected. Found atrophy of upper half of kidney with perinephritis. *Result*. Recovered.

Barnetson, J. (South Africa, 1947): Middle aged Negress. Found at autopsy. *Pathology*. Aneurysm 2 by 2 cm. just proximal to division of artery, right kidney. Tuberculosis of the wall of the aneurysm. Tuberculous bronchopneumonia and tuberculomata of heart.

Marrone, L. V. (New York, 1947): A 44 year old female with nine children. *History*. Pain in right flank and right upper quadrant for three years. *Examination*. Blood pressure 160/88. Wassermann; negative. Roentgenogram: Wreathlike shadow medial to right kidney. *Diagnosis*. Aneurysm of renal artery. *Treatment*. Nephrectomy, lumbar. *Result*. Cured. *Pathology*. At bifurcation of renal artery 2 by 1.5 by 1.5 cm. calcified aneurysm.

Puigvert Gorro, A., and Cols Baques, A. (Spain, 1947): A 38 year old male. *History*. For four years dull pain in right lumbar region. Two years ago passed ureteral stone. Pain continued. *Examination*. Could palpate lower

pole of right kidney. Tenderness in right renal area. Urine: Many pus cells; few red cells. Wassermann: negative. Roentgenogram: two dark shadows; one in parenchyma and the other in region of the pelvis. Left kidney not observed on roentgenograms. *Diagnosis.* Stenosis of pelvo-ureteral junction, right, and secondary stones. Absence of left kidney. *Treatment.* Proposed doing uretero-pelvic repair. Lumbar incision. Found small pulsating mass, size of a large pea on renal artery in front of renal pelvis. Was a fusiform dilatation of a terminal branch of the renal artery; this was excised. Removed stone and was unable to remove other stone because of hemorrhage. Did ureteropelvic anastomosis. *Result.* On second day other stone came out through the drain. Cured. *Pathology.* 7 by 8 mm. aneurysm.

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VAGOTOMY FOR PEPTIC ULCER

A Comparison of Results with Those of Gastric Resection

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THE surgical treatment of peptic ulcer has been controversial since Dragstedt first proposed the supradiaphragmatic section of the vagus nerves in the treatment of duodenal ulcer in 1943. Experimental work by Benjamin Brodie, showing the influence of the vagus nerves on the secretion of the stomach, was first done in 1814 and subsequent workers utilized varying degrees of vagus section in the treatment of peptic ulcer, including Latarget in 1922, Schiassi in 1925 and Pieri in 1932. The complete vagotomy for peptic ulcer and its popularization must be attributed to the work and influence of Dragstedt.

Not only has this subject been controversial in regard to the relative merits of vagotomy and gastric resection but also in the indications for vagotomy and in the technic. There have been certain workers who favored supradiaphragmatic vagotomy alone and others who preferred infradiaphragmatic operation with or without an associated drainage procedure. Because of this controversy we have reviewed the literature and compared a total of 60 cases of vagotomy of our own with 45 gastric resections in an attempt to assess the trend in regard to the present status of vagotomy for peptic ulcer.

In the first year after vagotomy was advocated by Dragstedt, there was a wave of enthusiasm and rush to try the procedure by surgeons all over the world. At that time the effects of vagotomy were not generally understood and the management of untoward side effects had not been clarified. Surgeons inexperienced in this operation probably also did a considerable number of incomplete operations. The results in general did not seem to be very satisfactory and there was a prompt denunciation of the procedure by a number of eminent and influential surgeons as well as by a considerable number of other less eminent surgeons. Following reports in the literature of poor results by such outstanding surgeons as

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Walters, Lahey, and others, many surgeons almost abandoned the procedure. Others, however, felt the operation held much promise and continued to work out some of the pitfalls. These were notably Dragstedt, Moore, Grimson, Crile, and others. As a result of their own and others' work, the reports coming into the literature now seem to be somewhere between the first swing of enthusiasm and the later pessimistic view. Among the workers reporting good results there has been a considerable divergence of opinion regarding the procedure of choice. Moore has advocated the extensive supradiaphragmatic procedure without any supplementary drainage procedure, while Dragstedt and his followers have recently been doing routine gastroenterostomies with infradiaphragmatic vagotomy. The Lahey Clinic has tried the combination of gastric resection and vagotomy but their results have been less satisfactory than those of gastric resection alone.

We found it difficult to establish a criterion for successful operation because patients in some instances had postoperative gastrointestinal complaints, although there was clinical and roentgenologic evidence of healing of the ulcer. Some patients, on the contrary, were pleased with their operation but later investigation showed the ulcer to be still present. In our series the results were recorded on the basis of the patients' statements that they were satisfied with the results, together with the objective clinical findings of weight gain, roentgenologic evidence of healing, and others. Any patient who had objective findings of ulcer as well as any patient who was not pleased with the results of the operation for any other reason was considered to be an unsatisfactory result. These criteria for the evaluation of the operation are a little different from those more often used which consist only of ascertaining whether or not the patient is free of ulcer findings and symptoms. They may, therefore, explain in part why our results were less satisfactory than those reported by other clinics.

Our cases were done by a number of different surgeons so that a variety of technics were employed. The majority of the cases were done by residents. There was a variety of types of operations which made tabulations of results rather complicated. We were, however, able to arrive at some rather definite conclusions.

Our 60 vagotomies were divided into six groups according to the type of procedure used. These were as follows:

Group 1. Supradiaphragmatic vagotomy only.

Group 2. Infradiaphragmatic vagotomy for gastric ulcer.

Group 3. Infradiaphragmatic vagotomy and gastric resection for gastric ulcer.

Group 4. Infradiaphragmatic vagotomy only for duodenal ulcer.

Group 5. Multiple procedures associated with vagotomy.

Group 6. Infradiaphragmatic vagotomy with gastroenterostomy for duodenal ulcer.

The patients' postoperative complications and complaints together with their clinical findings are shown in chart I.

The first group consisted of 1 case which was a supradiaphragmatic vagotomy for a penetrating gastric ulcer on the lesser curvature. This was done two and one-half years ago and the patient was well satisfied (chart I).

The second group consisted of 4 infradiaphragmatic vagotomies for gastric ulcer (chart I). Of this group, 3 were satisfied and 1 was not satisfied. The unsatisfied patient continued to complain of epigastric symptoms such as pain and bloating. Gastrointestinal series revealed delayed emptying of the stomach. Postoperative Hollander insulin test revealed that the vagus pathway may still be intact.

The third group consisted of 1 infradiaphragmatic vagotomy and gastric resection for a large gastric ulcer (chart I). This patient was well satisfied.

The fourth group consisted of 3 infradiaphragmatic vagotomies only for duodenal ulcer (chart I). Of this group only 1 was satisfied and the other 2 were not satisfied. Of the 2 patients who were not satisfied 1 patient still had epigastric pain and continued to lose weight. The second patient stated he could eat no better and continued to have epigastric symptoms. Postoperative insulin tests on both of these patients revealed that vagus nerves may still be intact.

The fifth group consisted of 14 multiple procedures in which vagotomy was associated with some other operation such as cholecystectomy, pyloroplasty, biopsy, jejunal diverticulectomy, or hiatus hernia repair (chart I). Of this group of 14 cases, 13 were followed and 7 were found to be satisfactory and 6 unsatisfactory. Of the unsatisfactory results in this group, 1 patient had a severe cardiospasm which gradually subsided. He continues to have pain and eats no better. The remaining 5 patients continued to complain of epigastric pain, loss of weight and bloating. Two of these patients had additional neuropsychiatric diagnoses such as psychoneurosis, mixed type, and constitutional inadequate personality with inferior intellectual insight. One of these patients had a vagotomy for a gastric and stomal ulcer following a previous subtotal gastric resection for a duodenal ulcer, without relief.

POSTOPERATIVE COMPLICATIONS AND COMPLAINTS IN THE UNSATISFACTORY GROUP OF PATIENTS

Vagotomy

Group	No. Cases	Satisfied	Not Satisfied	Case No.	Subjective Findings	Objective Findings	Postop. Hollander Test	Complications
I	1	1						
II	4	3	1	35	Pain in area of incision where costal cartilages were cut. Has gas and bloating.	X-ray—no ulcer defect. Delayed emptying time.	Pos.	Pain in incision area—costal cartilages cut.
III	1	1						
IV	3	1	2	3	Pain, nausea	Gained weight Vomiting	Pos.	None
				27	Pain, nausea Eating no better	Lost weight	Pos.	None
V	14	7	6	42	Pain, nausea	Jaundice, diarrhea Vomiting X-ray—no ulcer		Jaundice and diarrhea
				38	Pain, nausea	Lost weight Vomiting Diarrhea	Neg.	None
				22	Pain, nausea	Gained weight Diarrhea		Esophageal cardiospasm
	13 Cases Followed			37	Pain	X-ray—active duodenal ulcer	Neg.	Reoperated—more vagus removed and patient still had pain. NP diagnosis—psychoneurosis, mixed type.

CHART I

Group	No. Cases	Satisfied	Not Satisfied	Case No.	Subjective Findings	Objective Findings	Postop. Hollander Test	Complications
V—cont'd				17	Pain, nausea	Vomiting, distention X-ray—24 hr. retention		NP diagnosis—constitutional inadequate personality with inferior insight.
				33	Weak, eating poorly	Lost weight, diarrhea X-ray—chronic ulcer	Neg.	Dehiscence
VI	37	25	6 (1 died)	4				Death following surgery—developed alkalosis, low chlorides, low potassium, hyperpyrexia and death. Autopsy report—pulmonary edema, congestion and flabby heart.
				41	Pain, nausea, fullness. States he is satisfied with operation.	Vomiting. X-ray—deformed duodenal bulb	Neg.	None
				58	Pain	Vomiting. X-ray—no evidence of ulcer		NP diagnosis—conversion hysteria and mental deficiency
	32 Cases Followed			21	Dizzy, weak. No pain, eats anything. States he is satisfied with operation.	Tarry stools. RBC 3,280,000 Hemat. 24. X-ray—deformed bulb. Gained weight.	Pos.	None
				1	Pain, nausea	Diarrhea, vomiting. Weight same.	Neg.	None
				7	Pain, nausea	Vomiting. Weight same.		Episode of partial obstruction at gastroenterostomy site.
				20	Pain. States he is satisfied with operation.	X-ray—chronic duodenal ulcer.		Jaundice

CHART I—(cont'd)

In our sixth group we had 37 cases of infradiaphragmatic vagotomies with gastroenterostomy, 32 of which were traced with 25 satisfactory results, 6 unsatisfied, and 1 death (chart I cont.). Of the 6 unsatisfactory cases in group 6, 2 patients stated that they were satisfied with the operation but 1 of these was rated unsatisfactory because he continued to have some pain and the other because he had some bleeding 30 months postoperatively. The remaining 4 patients continued to have epigastric pain, nausea, vomiting and loss of weight. One of these 4 had an additional neuropsychiatric diagnosis of conversion hysteria and mental deficiency.

The one death occurred in a 34 year old white male who had severe obstruction from a duodenal ulcer and had been vomiting continuously for the week prior to entrance into the hospital. Following surgery he developed anuria, alkalosis, disturbed blood chemistry and finally hyperpyrexia with death. Autopsy findings revealed acute cardiac failure and pulmonary edema.

Of the 32 cases traced in this group which ranged from three months' to four years' duration, there were, therefore, 78 per cent good results (chart II).

Of the over-all group of vagotomy cases comprising 60 cases, a total of 54 were traced and of this number there were 38 satisfactory, 15 unsatisfactory and one death. A total of 70.4 per cent good results with a mortality rate of 1.6 per cent was therefore present in the over-all group.

A total of 19 of 23 cases of gastric resection for gastric ulcer were followed with 16 satisfactory, 1 unsatisfactory and two operative deaths (charts III, IV). The patient with the unsatisfactory result continued to have pain and fullness after meals, with no gain in weight. He continues to eat six meals a day.

Both of the patients who died had severe heart disease. One of these developed auricular fibrillation under anesthesia and surgery was cancelled on one occasion. A few days later he was operated on and his condition during the operation was fair. Following surgery he developed a mechanical obstruction which required reoperation and he died that same day. Autopsy findings revealed pulmonary edema and localized peritonitis.

The second patient had arteriosclerotic heart disease with a loud systolic murmur. During surgery his condition was only fair and he died on the table while bronchoscopy was being performed at the completion of the operation. The autopsy findings showed pulmonary edema, coronary arteriosclerosis, patchy myocardial fibrosis and chronic rheumatic mitral valvulitis.

POSTOPERATIVE COMPLICATIONS AND COMPLAINTS IN THE SATISFACTORY GROUP OF PATIENTS

Vagotomy

(The complaints in this group were transitory)

Group	No. Cases		
I	1		
II	4	Pain in incision—2 Pneumothorax—1 Dehiscence—1	Thrombophlebitis—1 Atonic stomach with gastric retention—2
III	1	Dehiscence	Peritonitis and incisional hernia
IV	3		
V	14 13 cases followed	Diarrhea—3 Foul eructations—1 Pain—3	Nausea—1 Vomiting—1
VI	37 32 cases followed	Pain—11 Vomiting—7 Nausea—7	Diarrhea—8 Painful wire suture—1 Thrombophlebitis—1 Atony, severe—1

CHART II

POSTOPERATIVE COMPLICATIONS AND COMPLAINTS IN THE UNSATISFACTORY GROUP OF PATIENTS

Gastric Resection for Gastric Ulcer

No. Cases	Satisfied	Not Satisfied	Case No.	Subjective Findings	Objective Findings	Complications
23	16	3 (2 died)	84			*Death*—Died on operating table during bronchoscopy after operation was over. Arteriosclerotic heart disease. Autopsy report—chronic rheumatic mitral valvulitis.
19 cases followed			105			*Death*—Secondary operation for obstruction 6 days after first operation. Arteriosclerotic heart disease Autopsy report—pulmonary edema with focal peritonitis.
			89	Pain, fullness after meals. Nausea.	No gain in weight. Still eats 6 meals a day.	None
				Gastric Resection for Duodenal Ulcer		
21	15	3 (2 died)	68			*Death*—Long difficult operation. Died on fourth postop. day. Autopsy report—acute peritonitis and necrotizing pancreatitis.
18 cases followed			75			*Death*—Operation done for massive bleeding. Condition critical throughout surgery. Died 2 days later. Autopsy report—acute pul. edema.
			81	Pain	Vomiting X-ray—Negative	Occasional episodes of vomiting. Readmitted to hospital as a mental case because of suicidal tendencies.

CHART III

POSTOPERATIVE COMPLICATIONS AND COMPLAINTS IN THE SATISFACTORY GROUP OF PATIENTS

Gastric Resection for Gastric Ulcer

(The complaints in this group were transitory)

No. Cases		
23	Pneumonia—1	Dumping syndrome—2
	Dehiscence—2	Severe hiccoughs—1
19	Diarrhea—2	Eviscerated and gangrenous loop of bowel resected—1
cases		Atelectasis—1
followed		

Gastric Resection for Duodenal Ulcer

21	Partial obstruction at gastroenterostomy site—2	Abdominal wall abscess—1
		Abdominal wall serum pocket—1
18	Evisceration—1	
cases	Mechanical obstruction reoperated—1	
followed	Disruption to peritoneum—1	
	Thrombophlebitis—1	

CHART IV

This gave 84 per cent good results and a mortality rate of approximately 8.7 per cent in the series of resections for gastric ulcer.

Eighteen out of a total of 21 cases of gastric resection for duodenal ulcer were followed with 15 satisfactory results, 1 unsatisfactory result and two deaths (chart IV).

The patient who was not satisfied continued to complain of epigastric pain and vomiting which was relieved by milk and cream. Repeated gastrointestinal series were negative. This patient was readmitted to this hospital as a mental case with suicidal tendencies.

Both of the patients who died had extensive gastrointestinal bleeding. One of these, in spite of extensive transfusions, could not be controlled, and surgery had to be done to stop the massive hemorrhage. Throughout surgery his condition was critical and he died two days later. Autopsy findings revealed acute pulmonary edema.

The other patient had a long, difficult operation, following which his condition continued to be critical. He died on the fourth postoperative day. The autopsy findings revealed acute peritonitis and necrotizing pancreatitis.

This gave 83 per cent good results and a mortality rate of 9.5 per cent. This mortality rate is extremely high compared with reports from the better clinics. In 2 of the cases, operation was done for massive hemorrhage, a condition in which a vagotomy would probably not have been feasible. But even if these cases were eliminated, the mortality rate of the over-all gastric resection group would be several times that of vagotomy.

The complications following vagotomy consisted of the following: wound dehiscence, thrombophlebitis, cardiospasm, gastric atony, pneumonia, atelectasis, diarrhea, dumping syndrome, foul eructations, jaundice and one death.

The complications following gastric resection for duodenal ulcer consisted of dehiscence, thrombophlebitis, mechanical obstruction with reoperation, abdominal wall abscess and serum pockets, partial obstruction at enterostomy site and two deaths.

The complications of gastric resection for gastric ulcer included: dehiscence, thrombophlebitis, pneumonia, atelectasis, diarrhea, dumping syndrome, severe hiccoughs, evisceration with a gangrenous loop requiring bowel resection and two deaths.

There were 2 vagotomy cases in which the vagus nerve was found to be still present at reoperation. Following his second operation, one of these patients was completely satisfied while the other has continued to be unsatisfied.

There were 2 cases who had had previous gastric resections and who continued to have ulcer complaints; both of these patients had a vagotomy and 1 was well satisfied while the other continues to have epigastric distress relieved by diet.

Three of the patients with unsatisfied vagotomy results had neuropsychiatric diagnosis of conversion hysteria, mental deficiency and psychoneurosis of mixed type, so that the true clinical results could not be clearly evaluated.

The Hollander insulin test was not done in a statistically significant number of cases. In general, it conformed with the clinical results, that is, where the insulin test showed complete vagus section, the results were better than when it was positive. This was not, however, universally true.

The percentages of satisfied and unsatisfied patients in the over-all group of vagotomies as well as in the group of vagotomies associated with gastroenterostomy is compared with the results of gastric resection in chart V.

	Followed	Satisfied	Unsatisfied	Postop. Deaths
Over-all vagotomy group	54 cases or 90%	38 cases or 70.4%	16 cases or 29.6%	1 or 1.16%
Vagotomy with gastroenterostomy	32 cases or 86%	25 cases or 78%	7 cases or 22%	1 or 2.7%
Gastric resection for gastric ulcer	19 cases or 82%	16 cases or 84%	3 cases or 16%	2 or 8.69%
Gastric resection for duodenal ulcer	18 cases or 85%	15 cases or 83%	3 cases or 17%	2 or 9.5%

Chart V

(Mortality rates based on the over-all numbers)

Discussion

We have reviewed 60 cases of vagotomy for peptic ulcer and compared them with 45 cases of gastric resection for peptic ulcer. Most of these cases were followed from three months to four years. Some of the early cases of vagotomy were done for gastric ulcer which, because of the high percentage of malignancy, is no longer advocated except perhaps in definitely benign (proved by biopsy specimens) high lying lesions, as is advocated by Dragstedt. Several of the cases of vagotomy were combined with other procedures not relevant to the ulcer, such as cholecystectomy or hiatus hernia. The operations were done by a number of different surgeons, the majority being done by residents, so that no uniform technic was used. For these reasons, the evaluation of the proce-

dure was made more difficult. We could conclude, however, that the vagotomy group which included gastroenterostomy seemed to give the largest percentage of satisfied patients. Even this group, however, had a lower percentage of good results than the gastric resection group. The mortality rate, however, was distinctly better in the vagotomy group and it is our feeling that the operation of vagotomy and gastroenterostomy can be done by the average surgeon with less mortality than the operation of gastric resection. Especially is this true in the more difficult cases and in the poorer risk patients.

Conclusion

After reviewing the literature and our own cases of vagotomy compared with gastric resection for peptic ulcer we have drawn the following conclusions:

1. Gastric resection is the opertaion of choice in gastric ulcer.
2. It is probably also the operation of choice in duodenal ulcer.
3. Vagotomy and gastroenterostomy may offer a special indication in technically difficult or poor risk patients.
4. When vagotomy is used, it should be done infradiaphragmatically and combined with gastroenterostomy.

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PHEOCHROMOCYTOMA COMPLICATED BY MULTIPLE DUODENAL ULCERS: A CASE REPORT*

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Up to the present time, over 210 cases of pheochromocytoma have been reported in the medical literature. Manasse[13] reported the first case in the literature in 1893. Following the classical description of the association of pheochromocytoma with paroxysmal hypertension by Labbé, Tinel and Doumer[12] in 1922, this condition has been reported in increasing frequency. Recently the diagnosis of pheochromocytoma has been facilitated by the introduction of the benzodioxane test, first suggested by Goldenberg, Snyder, and Aranow,[8] in 1947, which has subsequently proved to be almost specific. To the best of our knowledge, no case of pheochromocytoma complicated by massive hemorrhage from multiple duodenal ulcers has previously been reported. A report of such a case follows:

This 59 year old white male entered the Fort Logan Veterans Hospital on March 5, 1950, after having lost consciousness, which condition, associated with generalized convulsions shortly before admission, was thought to have been caused from an insulin reaction.

Past history revealed that this patient had been diagnosed as having had diabetes mellitus in 1941 when he developed typical symptoms of polyuria, polydypsia, polyphagia and weight loss. He was shortly thereafter started on 10 units of protamine zinc insulin per day, which dosage was continued until January 1950. In this month he was hospitalized at another veterans facility for weight loss and his previous diet was increased to 3000 calories. His insulin dosage was increased to 25 units of protamine zinc insulin and 10 units of regular insulin in separate injections each morning. It was one month after discharge from that previous hospitalization that the patient entered the Fort Logan Veterans Hospital.

The patient presented other significant factors in the past history; *i.e.*, symptoms of profuse sweating, which started about the time of his diabetic symptoms, episodic in character, and finally the chief factor which caused him to quit his work as a clothing salesman in 1948. The sweating was associated with blueness and coldness of his hands. The symptoms were investigated in another veterans hospital in 1944 for five months, but the etiology was not

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determined. He had also been considered for thyroid surgery in 1943 because of these complaints and an elevated basal metabolic rate.

Physical examination on admission revealed a poorly nourished, asthenic white male who weighed 125 pounds and who was 6 feet 1 inch in height. On admission he was mentally confused and disoriented, but shortly after ingestion of milk and orange juice, this mental confusion disappeared. The blood pressure was 160/110, the pulse was 90 and regular. The hands displayed a bluish, mottled cyanosis and were quite cold; the toes showed these signs to somewhat less extent. The skin overlying the phalanges was also atrophic in appearance and the nail beds were white. There was also a marked kyphosis of the upper dorsal spine.

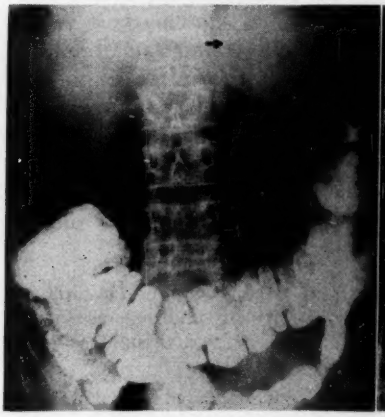

Fig. 1. Barium enema revealing large space-occupying lesion in the left upper quadrant.

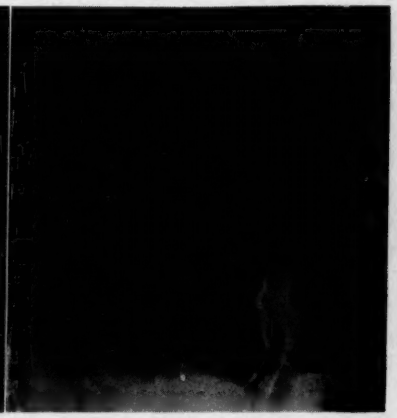

Fig. 2. Intravenous pyelogram illustrating inferior and lateral displacement of the left kidney.

Laboratory findings on admission revealed an initial blood sugar of 41 mg. per 100 cc., which rose to 328 mg. per 100 cc. six hours later, and thereafter, without insulin, ranged from 153.8 mg. per 100 cc. to 165.7 mg. per 100 cc. (fasting). The red blood cell count was 5,200,000. The white blood cell count was 15,400 with 62 neutrophiles per cu. mm., 37 lymphocytes, and 1 eosinophile. The urine revealed a specific gravity of 1.015, a 2 plus albumin, a negative sugar, and microscopically contained 12 to 14 white blood cells, 1 to 2 red blood cells, and 8 to 10 fine granular casts/h.p.f. Repeated white blood cell counts in the peripheral blood ranged from 13,400 to 16,200, all with normal differentials. Daily 24 hour urine specimens tested for quantitative amounts of glucose ranged from negative amounts to 18.4 Gm., but were usually below 10.0 Gm. The serum calcium ranged between 6.9 and 7.8 mg. per 100 cc. The serum phosphorus was initially 2.0 mg. and ranged between 1.4 to 4.88 mg. per 100 cc. The acid and alkaline phosphatase were 0.4 and 4.6, Bodansky Units, respectively. A barium enema revealed evidence of a large mass in the left upper quadrant of the abdomen in the region of the left kidney and the spleen (fig. 1). An intravenous pyelogram revealed this same mass and evidence of the left kidney being displaced downward slightly (fig. 2). This same shadow was also seen on a gastrointestinal series to deform the

lesser curvature of the cardia of the stomach. Also noted was dilatation of the first and second portions of the duodenum suggestive of a superior mesenteric artery syndrome. Roentgenograms of the flat and long bones showed marked osteoporosis with collapse of the bodies of several thoracic vertebrae.

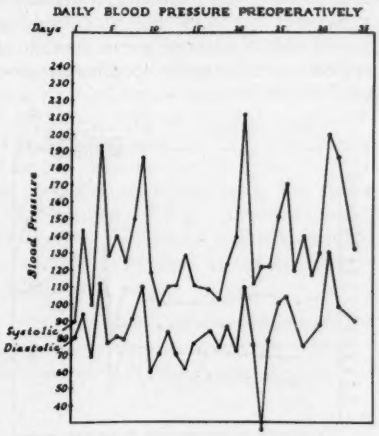

Fig. 3

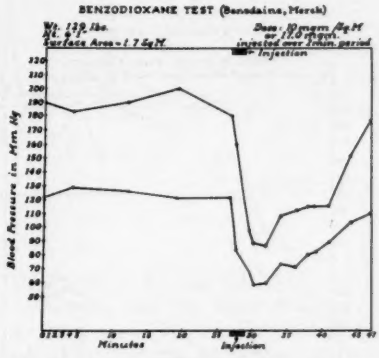

Fig. 4

The patient was admitted to the hospital with definite evidence of a hypoglycemic reaction which responded adequately to glucose ingestion. That same day it was noted that the patient was sweating profusely and, with the consideration of another hypoglycemic reaction, a blood sugar was taken, revealing 328 mg. per 100 cc. Because of the presence of hypertension on admission, daily blood pressures were taken by the nurses and found to vary from 90/50 to 250/140 (fig. 3), and one astute nurse had noted the presence of extreme coldness and cyanosis of the hands and feet, and profuse sweating whenever the pressure was elevated. These observations, plus the fact that the patient had been given a 5000 caloric test diet without insulin, and spilled very little sugar in the urine, led us to suspect very strongly a diagnosis of a

chromaffin cell tumor producing intermittent epinephrinemia. A barium enema taken for study of recent constipation had revealed a questionable shadow in the left upper quadrant, which showed up also on gastrointestinal series and intravenous pyelogram. A benzodioxane test was done at a time when the blood pressure was elevated, showing a marked drop in blood pressure (fig. 4). An intravenous histamine test was also done, when the blood pressure was normal, producing a marked rise in the blood pressure (fig. 5). With this clinical evidence, a diagnosis of pheochromocytoma in the left adrenal area was made.[3-7,10,11,15,16]

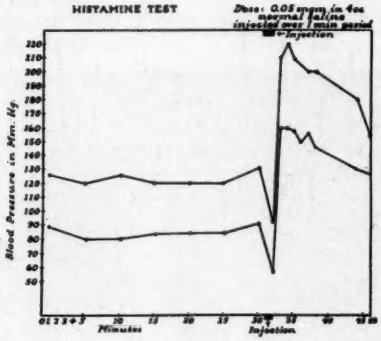

Fig. 5

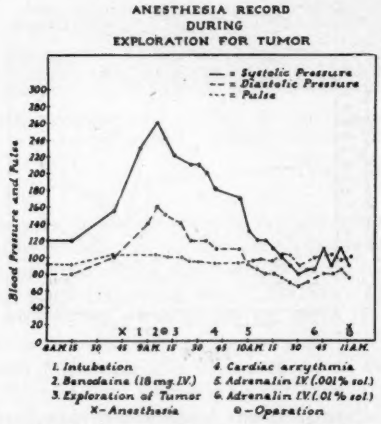

Fig. 6

On March 31, 1950, the patient was transferred to the Surgical Service, where all of the surgery discussed in this case report was performed by Dr. Paul M. Ireland, Chief of Surgical Service. Following transfer, his blood pressure continued to fluctuate, as has already been shown in fig. 3.

On April 4, 1950, excision of pheochromocytoma was carried out through a combined thoraco-abdominal incision under general endotracheal anesthesia. After the induction of the anesthesia and just prior to making the incision,

the blood pressure rose to 260/160. He was immediately given Benodaine (Merck), 18 mg. intravenously. The blood pressure gradually dropped to 100/80 and was maintained at that level with a continuous intravenous drip of a solution containing 0.01 per cent epinephrine in 5 per cent glucose in distilled water. Oddly enough, the blood pressure did not rise with manipulation of the tumor during surgery (fig. 6). The right adrenal area was first palpated and no evidence of tumor was noted. A large solid tumor approximately 8 cm. in diameter was removed from the region of the left adrenal gland. The tumor was intimately related to the left adrenal gland, approximately one half of which was of necessity removed with the tumor. The spleen was found to be about one half of normal size and was sharing a major part of its blood supply with the tumor. The incision was closed in the usual manner with one chest drainage tube being left in the left lateral eighth intercostal space. He received 1500 cc. of blood intravenously during surgery. He withstood the surgery fairly well and was returned to the surgical ward in fair condition. Subsequent report by the pathologist revealed the tumor to be a typical paraganglioma.

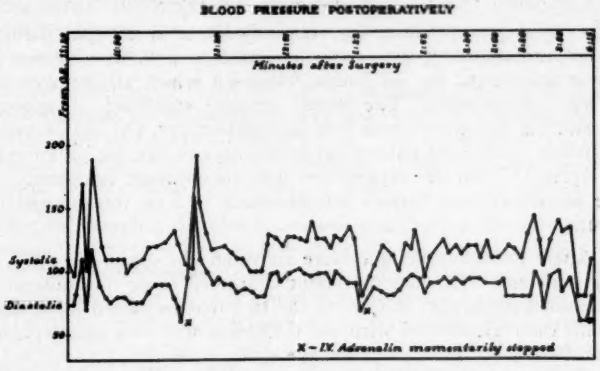

Fig. 7

Immediately after operation he was placed in an oxygen tent, an indwelling catheter was instituted, and the chest drainage tube was connected to dependent water seal drainage. He was given fluids ad lib by mouth. The intravenous solution of epinephrine in glucose started in surgery was continued (fig. 7).[6] In an attempt to reduce the amount of intravenous solution necessary to maintain the blood pressure, he was also given epinephrine in oil 2 cc. intramuscularly twice daily, and adrenal cortical extract 2 cc. intramuscularly every four hours. In spite of these measures, it was necessary to give the patient a total of 235 mg. epinephrine intravenously in the above stated solution during the first 24 hours postoperatively. The blood pressure, however, was maintained between 100-110/60-70, except for transient fluctuations when, of necessity, the intravenous bottles had to be changed (fig. 7).

On the first postoperative day, the patient was seen in consultation by two internists who felt that he presented the clinical picture of adrenal cortical insufficiency and recommended intravenous potassium chloride and calcium gluconate, and advised that the adrenal cortical extract should be increased. The blood calcium and potassium levels were determined to be within the

limits of normal. He was given 2000 cc. of 0.2 per cent potassium chloride in 5 per cent glucose in distilled water containing 20 cc. of 10 per cent calcium gluconate intravenously during the first postoperative day. To this solution was also added epinephrine so that he received 225 mg. intravenously during the day, in spite of the fact that the adrenal cortical extract was increased to 5 cc. intramuscularly every two hours. He was also given 1000 cc. of whole blood intravenously the same day. On the first postoperative day he was taking fluids well by mouth and complained only of an occasional abdominal cramping pain. His condition seemed satisfactory. An electrocardiogram revealed a complete right bundle branch block which completely cleared up by the following day and subsequent electrocardiograms revealed only evidence of electrolyte imbalance.

On the second postoperative day, it was possible to maintain the blood pressure at a satisfactory level, using a continuous intravenous solution containing only 0.005 per cent epinephrine. This was continued for the next 24 hours. A roentgenogram of the chest had revealed the chest drainage tube to be lying against the right diaphragm, which explained why very scanty drainage had been obtained, and the drainage tube was removed.

On the third postoperative day, the concentration of epinephrine in the continuous intravenous glucose was decreased to a 0.001 per cent solution which was maintained for 10 hours, following which all intravenous epinephrine was discontinued. The blood pressure stabilized at approximately 110/80 and his condition seemed to be satisfactory. On the following day, it was possible to keep the patient out of the oxygen tent for prolonged periods and on April 9, 1950, the oxygen tent was discontinued completely. On this day, the adrenal cortical extract was decreased to 5 cc. intramuscularly every four hours.

The patient gradually became more active and by April 11, 1950, was ambulatory with aid. His chief complaint continued to be intermittent generalized abdominal distention, in spite of the fact that he passed large amounts of flatus with ease. Associated with the distention was occasional epigastric distress and nausea. In an attempt to relieve the distention, prostigmine, urocholine, and belladonna were all subsequently tried, to no avail.

On April 15, 1950, it was noted that the patient had developed a left hydrothorax. Thoracentesis was carried out and 950 cc. of a rather viscid dark amber fluid was obtained. Subsequent chest roentgenograms showed progressive regression of the residual fluid.

A barium enema was done on April 19, 1950, and revealed no abnormalities except for moderate distention of the descending loop of the duodenum. It was felt that this was probably due to a so-called superior mesenteric syndrome which had been suspected on previous upper gastrointestinal roentgenograms. This syndrome was not confirmed at the time of surgery, as it had been felt best to avoid any unnecessary exploration which would prolong surgery. Sigmoidoscopy was carried out on April 22, 1950, at which time no abnormalities were noted. The adrenal cortical extract was decreased to 2 cc. intramuscularly four times a day and his blood pressure remained stable within normal limits.

Upper gastrointestinal roentgenograms were to be done on April 24, 1950. However, starting about 4 a.m., the patient had three copious loose stools in a relatively short period. He had not noticed the presence or absence of blood in the stools. At 8 a.m., he was noted to be in shock, with a blood pressure of

45/20. A rectal digital examination revealed fresh blood and an immediate blood count revealed 2.9 million red blood cells per cu. mm. Whole blood transfusions were instituted immediately and were maintained continuously. He was placed in an oxygen tent and was given nothing by mouth. He was not nauseated and did not vomit. During the day he had several copious stools of practically unadulterated fresh red blood. The blood pressure varied between 60 and 90 systolic. Adrenal cortical extract was increased to 5 cc. every four hours and he was also given massive doses of vitamin K intravenously.

By 4 p.m. the following day, the red blood count was up to 3.3 million and the blood pressure was up to 110 systolic. At that time the patient was taken to surgery where he promptly vomited a large amount of fresh red blood, which was our first good clue that the source of the hemorrhage was probably high in the gastrointestinal tract. Abdominal exploration was then carried out under endotracheal general anesthesia. A large posterior duodenal ulcer was found in the first part of the duodenum which had penetrated into the pancreas. There was a sclerotic open artery in the base of the ulcer. A partial gastrectomy, partial duodenectomy, and gastrojejunostomy were carried out. The patient withstood the surgery fairly well. Postoperatively a Miller-Abbott tube was passed and continuous suction was maintained.

Early in the morning of April 26, 1950, the blood pressure dropped and intra-arterial infusion of whole blood was instituted to augment the intravenous blood. This was discontinued at 9 a.m. The blood pressure was maintained at about 75 systolic during the remainder of the day. He continued to have occasional stools consisting of fresh red blood.

The following morning the blood pressure was around 90 systolic. At 10 a.m., he began to have frequent copious stools of fresh red blood and it was obvious that the gastrointestinal hemorrhage had not been stopped. He was rapidly digitalized because it was feared that he was on the verge of heart failure. At 4 p.m., he was again taken to surgery, where exploratory laparotomy was again carried out. Three new ulcers were found at the junction of the first and second parts of the duodenum, with a spurting artery in the base of one of them which was tied off. The ulcers were resected and a subtotal gastric resection was carried out. A cholecystostomy was also done, as it was feared that the opening of the common bile duct might have been compromised in the resection of the duodenal stump. The blood pressure dropped to 60 systolic during surgery and rose to 90 systolic at the end of surgery, with the use of intra-arterial transfusions of blood.

Postoperatively, the patient was in poor condition, never regaining full consciousness, and having loud moist rales in both lung bases. The red blood cell count was reported at 10 p.m. to be 5.39 million, and it was apparent that the bleeding had probably been controlled. However, the blood pressure suddenly dropped, the patient stopped breathing, and shortly after, heart action ceased. He was pronounced dead at 11:45 p.m. on April 27, 1950.

Autopsy the following day revealed nothing in addition to the findings at the time of surgery. The nodule in the region of the thyroid gland which had been suspected of being a parathyroid tumor, as an explanation for his generalized skeletal decalcification and hypocalcemia,[2] proved to be a benign thyroid adenoma. The cause of death was felt to be pulmonary edema and shock secondary to repeated extensive surgical procedures and massive gastro-

intestinal hemorrhage. The bodies of several vertebrae were found to be extremely osteoporotic and could be crumbled in the hand easily.

Discussion. It is interesting to note that, with no symptoms referable to the upper gastrointestinal tract prior to surgery, a high of 16 units of free hydrochloric acid in a fasting individual on response to the subcutaneous injection of 0.5 mg. histamine, and negative upper gastrointestinal roentgenograms except for a possible superior mesenteric syndrome, multiple duodenal ulcers were demonstrated both at surgery and at autopsy. That one of these ulcers was apparently chronic and three were very recent is also interesting to note. The possibility that the chronic ulcer might be etiologically related to chronic intermittent sympathetic stimulation due to the pheochromocytoma must be considered.[18,19] It has been experimentally shown that adrenalectomized animals almost routinely develop duodenal ulcers.[17] Clinically it is well known that the patient with Addison's disease has upper gastrointestinal complaints and is prone to develop duodenal ulcers.[17] This patient presented the clinical picture of acute adrenal insufficiency following removal of the pheochromocytoma along with the major part of the left adrenal gland. The association of duodenal ulcers with pheochromocytoma both pre- and postoperatively seems logical to us and it is only the absence of any previous report in the medical literature which seems unusual. Although moderately large doses of adrenal cortical extract were given postoperatively, even larger doses of the extract might seem to be indicated, or even more effective might have been the administration of ACTH or cortisone.

On admission, the serum calcium was found to be 7.8 mg. per 100 cc. and the serum phosphorus was 2.0 mg. per 100 cc. It was noted that the patient had considerable dorsal kyphosis and subsequent roentgenograms of the skeleton revealed generalized osteoporosis. Repeat serum calcium determinations ranged between 6.9 and 10.0 mg. per 100 cc., and the serum phosphorus ranged between 1.4 and 4.88 mg. per 100 cc. Normal acid and alkaline phosphatase determinations were reported. The presumptive diagnosis was osteomalacia. Two etiologic factors were considered.

In retrospect it can be assumed that, because the patient had some disturbance in gastrointestinal function due to a chronic duodenal ulcer, he might have had some decrease in absorption of calcium from the gastrointestinal tract. A more likely etiologic factor may have been the overproduction of ACTH due to the excess of a circulating epinephrine-like substance produced by the pheochromocytoma, causing a modified Cushing-like syndrome.[1,17] The adrenal cortical hyperactivity in this syndrome produces an excess of the "S" hormone at the expense of the "N" hormone, resulting in negative nitrogen balance with loss of protein-bound calcium and the protoplasmic matrix of the skeleton.[2,9]

It was necessary to give this patient intravenously a very large amount of epinephrine continuously for the first 58 hours following the removal of the pheochromocytoma in order to maintain the blood pressure within the range of normal. At the same time, moderate amounts of adrenal cortical extract were also given. In our review of the literature, no case was found in which the blood pressure had to be artificially maintained for so long a period postoperatively. Biological assay of the tumor was carried out by Dr. E. B. Pratt and Dr. Joseph H. Holmes of the Medical Department, with the assistance of Dr. Robert Grover and the Department of Physiology of the University of Colorado School of Medicine, according to the method of Mortell and Whittle (chart 1).[14] From this assay it was determined that the tumor, which weighed

230 Gm., contained approximately 10 mg. nor-epinephrine, or equivalent, per Gm. of tumor tissue, or a total of 2,300 mg. of the adrenaline-like substance.

CHART I

	Systolic B.P.	Diastolic B.P.	Pulse Rate
Control level	157	95	140
After injection of 10 gamma of 1-nor-epinephrine	210	129	100
Control level	135	88	140
After injection of 1 cc. of 1/1000 tumor extract	178	133	100
After injection of 1 cc. 1:1000 extract of skeletal muscle	No appreciable rise in B.P. or Pulse Rate.		

Biological Assay of Tumor Extract
(Assay done in a female dog weighing 10¾ kilo.)

In a recent case report, Cattell[3] states that the Trendelenburg position is all that is necessary to avoid cerebral anoxemia associated with the hypotension following removal of the tumor. It would be interesting to know what the biological assay of the adrenaline-like substance in the tumors he was reporting showed. It seems illogical to us to rely on the Trendelenburg position alone when the blood pressure is unobtainable by the usual methods, and the intravenous injection of a very small amount of epinephrine will rapidly restore the blood pressure to normal.

During his acute gastrointestinal hemorrhage, this patient received a total of 64 pints of whole blood, part of which was given intra-arterially, with the majority being intravenously, all within a period of 84 hours. This may or may not be some kind of a record, but is reported to stress the fact that during the administration of all of this blood the patient never had any signs or symptoms that could be attributed to a transfusion reaction.

Summary

1. A case of pheochromocytoma with surgical resection followed by fatal massive gastrointestinal hemorrhage from multiple duodenal ulcers has been presented.

2. Possible etiological factors in the production of multiple duodenal ulcers in this type of case have been discussed.

3. Some possible mechanism in the production of such an imbalance in calcium metabolism as manifested in this patient has been presented.

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PANCREATITIS: CURRENT CONCEPTS

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Acute inflammatory diseases of the pancreas have been known as pathological and clinical entities since the middle of the last century. These lesions were recognized sporadically at autopsy and, when symptoms were severe, at operation. But then, as now, acute pancreatitis could be only suspected, not diagnosed, by clinical manifestations alone. The desirability, therefore, of a specific pathognomonic indication of the presence of such disease was obvious. In 1909 Wohlgemuth[1] reported increased diastatic activity in the urine in acute pancreatic disease, and following this the possibility inherent in determination of pancreatic enzymes in blood and urine seemed promising. Effective realization came with the method of blood diastase estimation reported from the laboratory of this hospital by Somogyi in 1934.[2] With the availability of an accurate, reproducible and rapid method for estimating blood and urine diastase it soon became apparent, following postmortem and operative correlation, that this procedure constituted a highly specific test for the diagnosis of acute pancreatic inflammation. Shortly following the publication of this method the true incidence of acute pancreatitis became apparent. Since 1934 we have seen over 200 patients at this hospital with acute pancreatitis; diagnosis, in all instances, has been based on the combination of abdominal pain plus an elevated blood or urine diastase. Due to increasing familiarity with the disease the frequency of its recognition has become greater. In recent years acute pancreatitis has constituted about 0.3 per cent of all hospital admissions. This hospital has been continuously active in investigating inflammatory disease of the pancreas and in the recent past has established a separate laboratory devoted to these investigations. Principal participants are Doctors C. J. Heifetz, L. A. Sachar, M. Somogyi and the authors. The purpose of this report is to present our current concepts of acute pancreatitis as based on our investigations, clinical experience and performance of more than 100,000 clinical diastase determinations.

The diagnosis of acute pancreatitis in the patient with abdominal pain rests upon the finding of an elevated blood or urine diastase

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(or lipase), or demonstration of the characteristic morbid anatomy. Surgery is not indicated in the therapy of acute pancreatitis, and it is therefore fortunate, from the diagnostic standpoint, that the blood and urine diastase are elevated in the early stage of practically all cases. With this importance attached to the determination of diastase, it behooves the clinician to be well acquainted with the behavior of this enzyme.

It has been repeatedly shown that blood and, consequently, urine diastase are elevated when the pancreatic ducts of experimental animals are obstructed. Presumably, then, it is ductal obstruction in clinical pancreatitis that gives rise to the elevated diastase. Experimentally, duct obstruction will produce only transient pancreatic edema and an elevated blood and urine diastase. There are many clinical hypotheses regarding the etiology of acute pancreatitis and the disease has been produced experimentally in a number of fashions, but a common denominator in all is the combination of ductal obstruction and some type of pancreatic trauma. There is no decisive evidence that either alone will produce pancreatitis. While the pathogenesis of clinical pancreatitis may be multifold, the etiology is probably a combination of duct obstruction plus pancreatic tissue injury.

The normal range of blood diastase in man is 80-150 Somogyi units. In a state of fixed health the level in the given individual is quite constant and is not subject to diurnal or postprandial fluctuation.[3] In acute pancreatitis the blood diastase usually rises above 1000 units. It has been our experience that the highest blood diastase is almost invariably the earliest specimen obtained, even though this may be less than an hour after the onset of symptoms.[4] It is also the rule that consecutive specimens show a fairly progressive decline so that the normal level is frequently reestablished within 24 to 48 hours. The height of the diastase and the duration of its elevation do not parallel the clinical severity of the attack. Furthermore, there are no characteristic differences in the diastase behavior in acute transient pancreatitis and acute pancreatic necrosis.

Elevated blood diastases may accompany, in addition to acute pancreatic inflammation, other abnormal states. The most common of these is retention of normally excreted blood constituents due to renal disease. Renal retention may give rise to blood diastase levels of as high as 1200 units. The other abnormalities with which elevated diastases may be associated—obstructive and inflammatory diseases of the salivary glands, carcinoma of the pancreas, and perforated duodenal ulcer, usually, but not always, posterior—seldom give rise to levels over 1000 units. In this connection, it should be

borne in mind that, among other conditions in which a blood diastase level of less than 1000 units is found, is subsiding pancreatitis. Blood diastase levels of 1500 units or more may be considered pathognomonic of acute pancreatitis. For an elevated diastase of less than 1500 units to be accepted as diagnostic of acute pancreatitis the other causes of diastase elevation must be eliminated. It is interesting to note that sometimes in azotemia there is a normal blood diastase with a markedly decreased urinary clearance of the enzyme.[4] It thus appears that in some azotemias diastase formation is depressed, or the enzyme is destroyed or sequestered in the body. Our group is currently investigating this phenomenon.

If renal retention is suspected in a patient with a high blood diastase level, the significance of this level may be quickly established by determining the enzyme activity in the urine. It was observed in this laboratory in 1937[5] that the urine diastase level, unlike the blood diastase level, fluctuates markedly, but that it is always higher than that of the blood in ratios between 2:1 and 6:1. The same ratios obtained with the elevated diastase levels of acute pancreatitis. Conversely, these ratios were found to be reversed in the presence of renal retention. More recently we have been determining the urinary diastase activity in terms of the hourly excretion rate (units excreted per hour). Urine to blood diastase ratios by this method of measurement are, again, greater than unity unless there be renal retention.

Further investigations have recently been made of the relationship of blood and urine diastase during the course of acute pancreatitis.[6] Blood and urine diastase bear no exact mathematical relationship during the acute disease, but their responses are roughly parallel. In all of the cases studied the maximal urine diastase (hourly excretion) was recorded almost simultaneously with the highest blood diastase. In the presence of normal renal function an elevated blood diastase induces an increased urinary diastase excretion in something less than two hours, probably in the space of minutes.

As has been indicated, determination of the urinary diastase excretion rate will quickly identify or exclude the factor of renal retention. Urinary diastase excretion rates alone may also serve as a diagnostic test of acute pancreatitis. While the studies are not yet complete, we can at this time state that a rate of 1200 units per hour or over is pathognomonic of acute pancreatitis.

Much has appeared in the recent literature concerning the response of the blood diastase to pancreatic stimulants and the employment of these responses as the basis for tests of pancreatic

function. As we have pointed out[7] such a test, to be valid, must fulfill one of three criteria: the stimulus must invariably cause the blood diastase of normal individuals to rise; the stimulus may cause no elevation of the blood diastase in normal individuals, but causes an elevation of the blood diastase in individuals in whom the main pancreatic duct is blocked; the stimulus may cause inconstant variations or elevations of the blood diastase of both normal individuals and those with disease of the pancreas, but occasionally in the latter it produces the pain of which the patient complains. Prostigmine, mecholyl, secretin, morphine sulfate followed by mecholyl and secretin were administered to four groups of individuals: (1) a control group, (2) patients with a history of acute pancreatitis (some recently subsided), (3) patients with carcinoma of the pancreas, and (4) patients with pancreatic calculi. Serial blood diastase determinations were made on these patients as well as on a group which had received no medication. The control group with medication and a control group without medication showed no difference in diastase fluctuation. In the other groups of patients there was observed no consistent rise in the blood diastase after administration of any of the stimulants, and none of the patients had their abdominal pain reproduced.

As we have mentioned, since 1934 there have been over 200 patients admitted to the St. Louis Jewish Hospital with acute pancreatitis. In this group there have been nine deaths; autopsy revealed acute pancreatic necrosis in 7, and an abscess of the pancreatic head and multiple liver abscesses in 1. Thus our mortality rate in acute pancreatitis has been about 6 per cent.

Although 50 per cent of our cases have been subject to recurrent episodes of acute transient pancreatitis, none have developed pancreatic insufficiency or calculi. The latter conditions are rare in our experience. Only 3 of our cases have developed pancreatic abscess.

The nonoperative diagnosis of acute transient (interstitial) pancreatitis rests upon the combination of abdominal pain and an elevated blood or urine diastase. Since there is no specific difference in the clinical manifestations or in the behavior of diastase in acute transient pancreatitis and acute pancreatic necrosis, the diagnosis of acute pancreatic necrosis rests upon demonstration of the morbid anatomy at operation or autopsy. There is nothing characteristic about the type of pain or physical findings in acute inflammatory disease of the pancreas. However, as a group, the degree and persistence of shock in acute pancreatic necrosis is more marked than in acute transient pancreatitis. Since we do not perform surgery for acute inflammatory disease of the pancreas, we cannot say with certainty whether a severe attack of "acute transient pancreatitis"

is just that or "acute pancreatic necrosis" unless, of course, the patient succumbs and subsequently comes to autopsy. One can make a diagnosis of acute pancreatitis with assurance in a patient with abdominal pain and a blood diastase level of over 1500 units, or a urine diastase excretion rate of over 1200 units. In the presence of abdominal pain and blood diastase level between 500 and 1500 units, a diagnosis of acute pancreatitis can still be made if the other causes of diastase elevation are excluded. It is to be noted that this differentiation will at times necessitate exploratory laparotomy for the exclusion of perforated peptic ulcer. The facts being what they are, good medical practice requires that any patient with acute upper abdominal pain have a blood or urine diastase determination.

The treatment of acute pancreatitis presents three aspects: that of the acute episode, that of the complications that may occur, and that directed toward interruption of recurring attacks.

The fact that surgical intervention has nothing to offer the patient with acute pancreatitis is now commonly accepted. We have been firmly of this opinion since 1938.[8] The conservative management of the acute episode is symptomatic, supportive and prophylactic. The prominent, and usually sole, symptom of acute pancreatitis is pain of some degree. Narcotic and parasympatholoytic drugs are commonly employed for relief. In respect to the former, our experience does not substantiate the belief of some that morphine exacerbates the pain of acute pancreatitis. In respect to the latter, while we have not used them extensively, we have no objective substantiation of the ability of banthine or atropine to ameliorate pain or shorten the course of acute pancreatitis. Supportive care is necessary for those patients in whom shock is manifest, or whose attacks are sufficiently marked and prolonged so that the nutritional requirements, in all components, cannot be met or are therapeutically diverted from the gastrointestinal tract. It is unnecessary here to give in detail the measures employable for shock and general supportive care. Prophylactic therapy of acute pancreatitis is aimed at decreasing the incidence of the complications of abscess and purulent peritonitis by use of antibiotics, and the severity and progression of the attack by depressing the secretory activity of the pancreas which may be accomplished by gastric suction, withholding oral food intake, and the administration of the antisecretory drugs.

Our experience has been that the majority of patients with acute pancreatitis will fully recover without the necessity of supportive care, and without employment of the prophylactic measures. When indicated, symptomatic and supportive care are, of course, essential.

The value of prophylactic treatment is not objectively assayable in our cases. Theoretically, the possible value of antibiotics warrants, we believe, their routine usage in acute pancreatitis. We do not routinely employ the antisecretory measures in pancreatitis. In most cases, however, we do prohibit oral ingestion until there is some clinical improvement.

Delayed treatment of acute pancreatitis is primarily surgical, and is indicated when complications, such as pseudocyst and pancreatic abscess, arise, or when disease of the extrahepatic biliary tract is evident. We shall not elaborate on the former.

The high association of calculous disease of the biliary tract has been well known since first pointed out by Opie.[9] There is no doubt, as indicated by Opie in his common channel theory that biliary calculi are frequently the basis of the pathogenesis of acute pancreatitis. It is also true, however, in our experience[10] and that of a great many others, that acute pancreatitis recurs in a distressingly significant number of patients following the surgical eradication of biliary tract disease. In the light of these various considerations, it is our policy to investigate routinely the biliary tract several weeks following subsidence of acute pancreatitis, and to recommend the surgical correction of any pathology found. On the other hand, we do not recommend primary or secondary biliary tract surgery in the absence of demonstrable pathology.

In that group of patients who have recurring episodes of acute pancreatitis and no evidence of biliary calculi, it has been rationalized that the pathogenetic mechanism of the common channel is nevertheless operative in many, the basic lesion being obstruction secondary to spasm of the sphincter of Oddi. On the basis of this the operation of sphincterotomy has been advocated for such patients. We have had insufficient personal experience with this form of treatment to draw any conclusions.

Knowledge of acute inflammatory disease of the pancreas is far from complete. Of first importance is elucidation of the pathogenetic mechanisms involved, and of the responses of body physiology to the disease process. When this is achieved, the relationship of the two forms of pancreatitis will be established, diagnosis and prognosis will become more accurate, and new approaches to therapy will be provided.

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THE CONSERVATIVE TREATMENT OF BACKACHE

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THE skillful utilization of rest, in the art of medicine, is often neglected because of its simplicity. Rest is the physiologic antagonist of fatigue, stress, traumatic insult, and degenerative encroachments. The induction of rest is the basis of most conservative measures in the treatment of the painful back. It is the key to restoring equilibrium. A great number of painful back cases are those people who know no limit to their endurance. There may be no organic disease or injury present, but, physical and mental effort beyond the endurance of a vulnerable structure, such as the low back, must eventually cause it to give way. Tenseness, anxiety, loss of sleep, and mental depression may magnify the distress. The patient often must be taught how to "live within the restrictions of his back."

Surgery is reserved for the more specific indications where the pathologic state is intolerable and irreversible by conservative means. Recognition of the herniated disk has been revolutionary and the surgical relief for this condition is spectacular. These facts, however, should not lead, too readily, to the surgical route. The average person with distressing back symptoms is all too ready to accept the favorable prospect of immediate relief through surgery. The persistence of symptoms, after operation, may lead to great disappointment. A waiting period, and the application of conservative measures, is usually highly advantageous in the selection of those cases that definitely need surgical treatment.

Diagnosis. Conservative therapeutic measures versus surgery depends greatly upon the accuracy of diagnosis. The diagnostic significance of back pain depends greatly upon the thoroughness in which a pertinent history is elicited and upon the application of exacting clinical tests, together with roentgenographic and laboratory investigations. The influence of disease and metabolic disorders and postural imperfections must be thoroughly considered. When the diagnosis is accurately established, the therapeutic battle in the treatment of backache is half won.

Physiology of Rest. The spinal structures in the upright human body are never at rest. Steindler[1] states: "The spine is a multiple articulated mechanical unit of the body, subject to the stresses of locomotive power and body activity. Since the articulations of the

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spine are within a curved, rather than a perpendicular line of gravity, it necessitates the exertion of a constant muscular force to oppose and hold the rotatory forces. The muscles of the back are always at work. The two intrinsic properties of muscle tissues in locomotion are elasticity and contractivity.

The elasticity to tension stresses depends upon the histologic structure, especially that of the sarcolemma and connective tissue-sheath enveloping the muscle fiber. The elastic fibers of the muscle cells provide the stretching and contracting characteristics. Atrophy produces a reduction of muscle cell content. Degenerative changes lower the intracellular energy. Exhaustion of muscles produces myositic reactions."

These factors all produce a greater resistance to elasticity. Loss of elasticity in turn reflects abnormal strain on the articular structures which eventually exhibit painful rebellion. Direct localized pain is usually understandable. Indefinite radiating pain, resulting from the reflex phenomenon and the influence of sympathetic nervous tension symptoms are often the source of confusion.

Conservative Measures. The essentials of conservative treatment are:

1. Rest.
2. Postural correction.
3. Physical therapy.
4. Medical supportives—Correction of underlying diseases or deficiencies which derange skeletal structures.
5. Exercises.
6. Manipulations.
7. Local anesthesia.
8. Roentgen ray treatments.
9. Psychiatric encouragement.

Induction of Rest. Rest must include the body as a whole, as well as the low back. There are numerous methods of inducing rest. There seems to be a lack of understanding of what each type of treatment can or cannot accomplish. The various essentials are:

(1) Body relaxation and sleep,
(2) traction,
(3) support or immobilization,
(4) postural correction and
(5) analgesics and sedatives.

Complete relaxation is obtained only through bed-rest and sleep. Related factors of body fatigue are thus rendered an excellent opportunity to restore physiologic equilibrium. The methods of bed-rest are important. The patient should be hospitalized if possible. It is all but useless to advise the patient to stay in bed at home unless he is so ill and exhausted that he cannot get out of bed. After the patient has been in the hospital and trained in the details of complete bed-rest, a similar effective routine often can be established at home. It usually requires from two to four weeks to conquer severe pain and muscle spasm. Herniated disk symptoms that do not respond should receive surgical consideration.

Beds. The hospital bed should be fully adjustable to permit flexion of the knees and hips. It should be equipped with traction bars at the foot for pelvic and leg traction, or bars and pulleys for head traction. The mattress should be firm.

The home bed, or ordinary bed, should be equipped with boards placed between the mattress and springs. Pillows can be used for knee flexion, or a lift placed under the distal two feet of the mattress to form a deck for the legs. If complete rest is ordered, there should be no bathroom or other privileges of being out of bed at any time.

Traction. The muscles of the back all tend to pull the joints together. There are no muscles to pull them apart. Ligamentous and muscular rigidity or contractures, articular compression, and irritation, are relieved by traction. Traction may be applied in bed or in ambulation. Traction in bed for the low back is best obtained through a canvas pelvis belt equipped with leg straps and attached to ropes and pulley, with about 15 pounds of weight for each side. Traction by means of adhesive plaster and Buck's extension to the legs is very unsatisfactory. Usually the bed is adjusted to flex the knees and hips at about 25 degrees. This pelvic traction is continued day and night, except for short periods of relaxation and time out for physical therapy treatments. The most common error is to discontinue traction too early. Muscle spasm and radiating pain should be well overcome before permitting ambulation.

The Sayre head suspension apparatus is used for cervical traction. The chin is well padded. There should be a crossbar wide enough at the head to prevent pressure about the face or ears. Ten to 15 pounds of weight may be used. Ruth Jackson[2] has devised a special contour pillow to be placed under the neck to support the concave contour of the cervical spine. In most cases an overhead trapeze can be supplied for the convenience of the patient in changing his

position. In some cases of severe osteoarthritis of the lumbar spine, traction in full extension is preferable to flexion.

Support and Immobilization. Ambulatory support, by means of bracing, must follow sound mechanical principles. A proper corset, or brace, is a restraint for the purpose of equalizing the stresses of equilibrium. The common practice of applying the so-called "sacroiliac belt" indiscriminately is to be deplored. Either the back needs support or it does not. There are many designs of efficient back supports. Paul Williams has stressed the advantages of plaster casing, or of a brace to produce lumbosacral flexion. Others prefer a brace with low back uprights and pelvic band, such as the Meyerding "rocking chair" back brace. Where more rigid immobilization is desired, a modified Taylor, or Knight spinal type of brace, is used. Plaster jackets are reserved for the cases requiring complete immobilization. The plaster jacket has somewhat lost its popularity. Before the day of surgery for the relief of herniated disks, however, it was quite common to resort to complete plaster immobilization to prevent painful movement in the lumbar spine. It is still good treatment. In order to completely immobilize the lumbar spine at least one leg, down to the knee, must be involved in a plaster spica. Williams[3,4] has described the flexion cast. Hauser[5] describes a corrective cast. Women object to the regular types of leather covered braces and abhor the plaster cast. They must be favored with a support of corset style that includes bracing principles. Stock low back girdles may be reinforced with metal uprights in the back to make satisfactory supports.

In the cervical region the means of support may range from the simple felt collar to any one of several designs of metal neck braces, with adjustments for traction. The simplest one is made of small round steel wire, with chin and occiput support. It is made removable and it is adjustable for traction by means of shoulder straps and crossbars resting on the chest and back. This brace is called the Marshal-Foster cervical brace. The Shrock collar is also a very useful, removable neck support. Braces are to be worn only for the period necessary to reverse the painful localized pathology. Overbracing results in atrophy, rigidity, and weakness, which oftentimes is more detrimental than the condition for which the support was applied. Caution should be used in the application of metal braces in compensation cases. They may be used as an excuse for continued disability.

Adhesive plaster has long been a dependable means of temporary support. It is adaptable especially to the acute, mild, lumbosacral strain. In severe acute cases complete hospital bed-rest should be ordered rather than to permit ambulation with adhesive plaster

strapping. Where adhesive plaster is used it should be applied in a manner that will definitely support the area of local pathology. Compound tincture of Benzoin previously applied to the cleansed skin will greatly prevent skin irritations.

Postural Correction. The spine is an elastic system of bones and ligaments which are kept in a perfect state of dynamic equilibrium in all positions it assumes. For perfect balance of equilibrium, all lines of gravity are passed through the centers of articulation. The articulated body must rest on the heel and ball of the foot. There is constant exertion of muscle force necessary to oppose and maintain the rotatory forces of articulations of the body. The countless jolts from twisting, jumping, riding, or lifting, are sustained by the spine without discomfort if it is normal.

Advanced age, adiposity, occupational hazards, physical impairments, and many other factors enter into the development of the postural state. In most people with good health, improper postural affects become adequately compensated. There is usually some background of injury or systemic weakness where symptoms arise.

The short leg should have a lift on the sole of the shoe. The scoliotic spine should be supported or braced. The adipose abdomen will require support in addition to support of the back. Stresses arising from postural faults of an occupational nature will require analysis and adjustment of working requirements. Also, kyphosis with age or osteoporosis accentuates the cervical and lumbar curves, thereby increasing strain. Postural attitudes, such as that of ironing, or that of a mother frequently lifting her child during the day, require rigid instructions as to how to prevent undue stress to the back. A small stool under the feet while sitting at work is usually a relief for postural stresses. Occasionally a small pillow under the buttocks on the site of lumbar pain will relieve the stress of spinal curvature. Persons who do heavy work should be taught how to lift from the squatting position. Overexertion of the spine at any time is harmful. The individual who has had low back pain should not be permitted to cary out working activities which require torsion stresses to the lumbar spine. The farmer, on his tractor, should not twist around to handle levers back of him. The laborer should lift his loads from straight before him and not from one side or the other.

Physical Therapy. Physical therapy methods are the more dependable therapeutic agents in the treatment of backache. At the same time, there is often overrated use of them, and abuse. The common practice of administering daily light treatments by an untrained assistant is simply a convenient disposition of the case for

its psychological effect. Definite physiologic reactions may be extremely beneficial if prescribed according to indications for the various modalities of physical therapy. Infrared, short-wave, hot fomentations, ultraviolet wave, and galvanism have their definite indications. Exposure of the tissues to heat results in increased capillary circulation and its increased metabolism. An ice pack kept continually on a painful point in the back may succeed where heat fails. Relaxation is an important reaction of heat. It is also an important preliminary to massage and exercise. Low grade inflammation is relieved through increased oxygen and nourishment brought to the localized areas involved. Fever therapy, by means of short-wave, immediately relieves acute myositis, especially that of the type of acute wry-neck, or acute "catch" in the back. The body temperature need not be elevated to more than 99 or 100 degrees over a period of one hour. Autohemotherapy, in which 20 cc. of the patient's own blood is injected back into the buttock every third day is of value in addition to fever therapy.

Medical Analgesics and Supportives. The medical treatment of back pain is indicated:

1. To relieve pain and to produce relaxation.
2. To retard or reverse disease processes.
3. To restore tone and equilibrium to the tissues.

Sedatives and narcotics are to be used only to subdue extraordinary pain, anxiety and restlessness in order that more constructive measures may become effective.

The diagnosis of rheumatism or arthritis in the presence of back pain should not be used promiscuously, apparent arthritic changes so predominantly present in the roentgenogram are indicative of slow progressive systemic faults, finally manifested by painful reactions and disability. Ishmael[6,7] has stressed the vulnerability of the joints to the menopausal state. He emphasizes the importance of distinguishing osteoporosis from osteomalacia. Either of these systemic phenomena may lead to the joint tissues succumbing to the constant forces of stress and trauma. The healing process, finally in evidence as a spurring and lipping of the ligaments and joint margins, is diagnosed osteoarthritis.

The steroids, wisely administered, will reverse the demineralizing effects of osteoporosis. Testosterone propionate, 50 mg. may be given weekly. Estrogens are given in dosages averaging 20,000 units weekly.

Osteoporosis may also result from malnutrition, such as hypo-

proteinemia, hypovitaminosis C, and the type associated with pellagra. A balanced diet and food concentrates are indicated.

Osteomalacia or adult rickets may also render the spine susceptible to stress. It is caused by malnutrition, gastrointestinal disease interfering with absorption of calcium, kidney disease resulting in acidosis, or secondary hyperparathyroidism. The calcium in the bone must be replaced by the administration of calcium and vitamin D, and the substitution of potassium for sodium salts will prove effective measures.

Exercises. Exercises are designed for specific purposes. Locomotive efficiency depends on the phenomena of fatigue, the ease with which it occurs, its persistence, and the manner in which the muscles recuperate from it. It is ordinarily the business of the circulation in the muscles to remove the metabolic products of muscular activity promptly and thereby open the way to recovery and renewed activity of the muscles. Exercise must be gently but definitely applied in regular rhythm and a habitual routine. The patient should be cautioned against overexertion. The exercises have two principal objectives:

1. The painful extensors of the back and tightened hip flexors are energized by the execution of stretching maneuvers.

2. The hamstrings, abdominals, and gluteals are activated to improve strength.

The following exercises were devised by Waldrop and Shorbe[8] and are used routinely in our clinic (figs. 1-6):

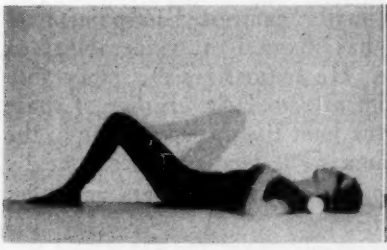

Fig. 1. Exercise No. 3

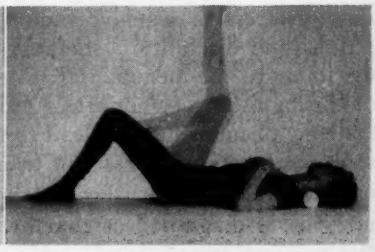

Fig. 2. Exercise No. 1

Exercise 1. On back: Bring the knee onto the abdomen; forcibly extend the knee, stretching the hamstrings, for a count of 10, then lower extended limb slowly. Relax and repeat five times for each leg, graduate to 25 times (fig. 2).

Exercise 2. On back: Feet on table, hips and knees flexed with pelvis held down flat; lift the lower lumbar spine up and down forcibly for a count of 10; graduate to 25 times (fig. 3).

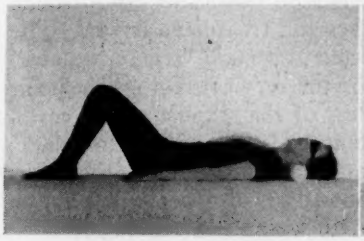

Fig. 3. Exercise No. 2

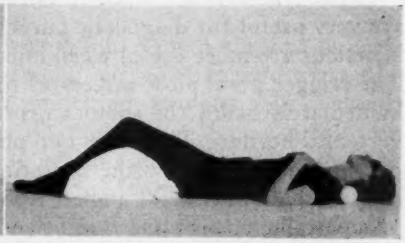

Fig. 4. Proper sleeping position

Exercise 3. In same position as No. 2: Force knees sidewise to touch table, right and left, for a count of 10 (fig. 1).

Exercise 4. In same position as No. 2. Draw one knee up on chest, then rest it and flex the other; then flex both knees on chest, using abdominal muscles (fig. 6).

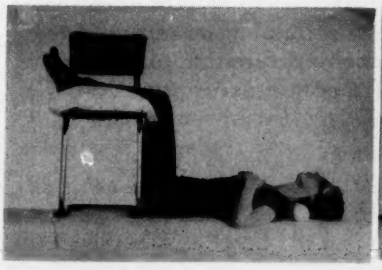

Fig. 5

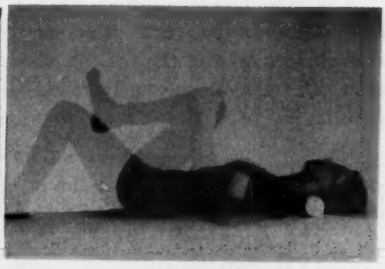

Fig. 6. Exercise No. 4

Fig. 5. Lying in this position is the best first aid for painful low lack. The contour pillow beneath the neck adds rest and relaxation, as well as improving posture. An electric heating pad may be placed beneath the low back.

Manipulation. Forcible manipulation of the spine is very effective in certain well selected cases. Before the surgical excision of herniated disks became popular, forcible manipulation, under general anesthesia, was used frequently in acute cases and immediate spectacular relief was obtained. One who attempts to use this method should master the various maneuvers thoroughly. Great caution should be used not to produce injury to rheumatic joints or aggravate a tubercular joint. The effectiveness of manipulation is that of breaking adhesions, and the stretching of contracted muscles and ligaments. If one is skillful enough to use the various holds and positions for specifically applied maneuvers, he may accomplish good results without anesthesia. Manipulations of the back under

anesthesia requires knowledge of special technic and the caution of experience.

Local Anesthesia. Local infiltration of novocaine or procaine are very useful for diagnostic purposes and treatment as well. The injections are more useful when injected at certain "trigger points." The trigger point most successful for the cervical region is a point immediately below the spinous process of the scapula at the costovertebral border. There are two points most effective in low back: one immediately below the posterior spine of the ilium, the other deep in the lumbosacral angle. About 10 cc. of 1 per cent solution of novocaine or procaine is sufficient for a single injection.

Roentgenotherapy. Roentgen ray is of principal benefit for the relief of acute pain due to chronic inflammatory reactions in degenerative arthritis. It should be used cautiously, and only as an initiative agent of relief.

Summary

The conservative treatment of back pain must rely chiefly upon the induction of rest, exercise, and medical measures to restore equilibrium to the tissues. A thorough appreciation of these measures over a long enough period of time will favor most gratifying results.

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PROBLEMS OF PROTEIN ALIMENTATION

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The importance of proper protein nutrition in surgical patients has been well recognized among surgeons for the past decade.[1,4,7,9,13,16] Adequate protein nutrition protects our patients against surgical shock, enhances resistance to infection, aids in repair of traumatized tissue, aids resistance to hepato-toxins and other injurious agents, and is essential in maintaining a normal mass of erythrocytes and plasma proteins.[2,4,5,6,9,18] It is well to recognize that the state of the patient's protein nutrition is a vital factor in deciding the success or failure of a major operation.[14,17,18] Many leading investigators have stressed the importance of proper protein alimentation in surgical patients,[1,4,7,9,13,16] but too few have acquainted us with the fact that there are problems of securing an adequate protein intake, nor have they informed us of ways of overcoming such problems. Ordinarily, if a patient has a good appetite, he is either in no need of high protein feedings or it presents no problem in this regard. Usually, the patient who is in serious need of forced protein intake, is debilitated, anemic, pessimistic, has no appetite, and will either only tolerate a slight increase in diet or, under forced feeding, will develop nausea, vomiting, bloating or diarrhea.

The need for high protein intake falls roughly into three categories: (1) those patients whom we are preparing for surgery who are debilitated through chronic disease, sepsis or malignancy; (2) those patients in whom, following surgery, it is necessary to limit or exclude entirely oral intake, or whose loss of protein is much greater than their intake; (3) patients who will require numerous surgical procedures, and in whom it is vital to keep their protein nutrition at a proper level to insure success of future surgical procedures. Each of the above categories has its own peculiar problems.

Those patients who are malnourished and whom we are attempting to prepare for surgery present us with the following:

They ordinarily are anemic, with a contracted vascular bed, and have low serum and body proteins; there is an atrophy of the gastrointestinal mucosa, and they have no appetite. These patients have adapted themselves to a low oral intake and forcible feeding is difficult. We should first correct their anemic state with transfusions to such an extent that their hemoglobin is above 80 Gm. per 100 cc.

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Thus, their protein intake will be used for augmenting body proteins rather than for producing red cells. It is important to realize that adequate nutrition in the debilitated patient cannot be established in a few days. To provide an adequate intake in these patients, it is necessary to follow some essential rules:[9,15] (1) one must impress on the patients the necessity for increased intake; (2) their idiosyncrasies must be given consideration; (3) body movement should be encouraged; (4) their diet should be complete and adequate; (5) sedation should be used sparingly and judiciously; (6) between meal feedings of liquid protein products should be given and, (7) the products and foods administered should be palatable. The doctor should survey their diet and advise a high protein diet of foods they like and will tolerate. Vitamins, particularly of the B complex, should be administered.

It has been observed by the author that the majority of commercial protein preparations used for supplemental feedings are distasteful even to a well person, and if forced on patients, they will become nauseated, lose their appetites, and the doctor will be defeated in the initial phase of his program.[8] It has been further observed that commercial protein preparations of a lower percentage protein are in general more tasteful, more soluble, and are, as a rule, better tolerated than the more concentrated products. Dry skim milk powder has 35 per cent protein, is inexpensive, and when made up in the proper formula, is agreeable and well tolerated. It has the qualities necessary for high protein supplementation. A formula used with good results is illustrated in table 1. It is wise

TABLE 1

High Protein Mixture for Oral Feedings

Dry skim milk powder	oz. 4½
Skim milk	qt. 1
Sugar	tsp. 1
Flavoring as desired	
May add 1 egg to formula	
Contains approximately 80 Gm. protein per quart	

to alternate products in a prolonged feeding program to avoid monotony and to keep the cooperation of the patient. Other low percentage products such as Protenum, Meritene, and Dietene, are well accepted by patients and may be alternated with dry skim milk in the same proportion, for this purpose.[8] One may obtain from 70 to 80 Gm. additional protein per day from these supplemental products without seriously impairing the patient's appetite. Two recent concentrated products, Stuart's Amino Acids and HPS-60, have

been well accepted by a small group of patients tested. The above mentioned procedures may be carried out for a period of time before the patient enters the hospital. In many instances, at the time of hospital admission, the patient's nutrition will still be below normal. If so, in addition to oral supplementation, we may give intravenous proteins. These should be given at night as they impair the patient's appetite. Five per cent alcohol added will give extra, much needed calories. Additional transfusions will frequently be needed.

The most difficult problems are encountered in the second category—patients in whom following surgery over a prolonged period of time, it is necessary to limit or forego entirely oral intake, or patients whose protein loss is much greater than their intake. The problems faced in this category are twofold: (1) how to obtain an adequate protein intake and (2) how to procure adequate calories. Intravenous proteins are not well utilized by the patient in the postoperative period,[11] but it has been aptly observed by Elman that a certain percentage of them is definitely utilized; thus, we are faced with the question of whether to be satisfied with a quarter or half a loaf, or none at all.[10,19] I ordinarily do not employ intravenous protein feedings until the fourth or fifth postoperative day. If, at such time, prolonged use of intravenous foods must be given, I routinely use them. A number of reports have been forthcoming of the value of intravenous alcohol postoperatively as a replacement of sedation.[12] I find its worth as supplying calories in patients in this category to be its greatest value. It may be used as a 5 per cent solution with 5 or 10 per cent glucose in the early postoperative phases and later may be given with intravenous proteins. I have also used 10 per cent glucose in Amigen for a week to 10 days in patients where additional carbohydrates were needed. This will also supply additional calories. If such solution is used, small needles and rotation of veins after each liter of solution should be employed to prevent thrombophlebitis.

In the third category are patients who will require numerous surgical procedures such as those with extensive trauma or burns, in whom we are faced with the problem of maintaining their nutrition at an adequate level for success of future operations. These patients are bedridden, pessimistic, have little appetite, and usually cannot be forced to eat. Their intake is far below their need. Their diet habits should be surveyed and they should be allowed to select a diet most pleasing to them. For this type of patient plastic tube feedings are invaluable. Patients do not tolerate prolonged indwelling, large rubber or plastic tubes. However, they suffer but little discomfort from a small plastic tube and can, in addition, eat their prescribed diet without difficulty. A problem encountered in this

type of feeding is a method of introducing it through the plastic tube into the stomach, as drip feedings cannot be used because of thickness of the preparation. Blocker[5] has obtained excellent results with pump feedings. However, it is somewhat difficult to adjust the proper rate of flow of this mechanical device. It is expensive, and it is not easily adaptable to the ordinary hospital. I have found that if a formula is made containing 2400 cc. and this is injected with a syringe at the rate of 200 cc. every two hours, we may obtain an adequate fat, protein, carbohydrate and caloric intake. A second problem we face in this type of feeding is unorthodox reactions of the patient's gastrointestinal tract. If too concentrated a product, particularly protein concentrates, is given too early in our feeding program, the patient may develop nausea, vomiting, bloating, or diarrhea, thereby defeating the purpose of our feedings. It is wise, therefore, to start with a bland preparation and gradually add the concentrated foods. Table 2 reveals

TABLE 2

Plastic Tube Feeding

	C.	P.	F.	Cal.
1st Day—Homogenized milk cc. 2400	120	72	96	1632
2nd Day—Eggs 2		12	12	156
3rd Day—Stuart's Amino Acids tbsp. 4		20		120
4th Day—Dextrimaltose Gm. 100	100			337
5th Day—Stuart's Amino Acids tbsp. 4		20		120
	220	124	108	2365

the regimen customarily employed for plastic tube feedings. If at any time during the feeding program the patient complains of bloating, nausea, or a feeling of fullness, it will be wise to revert to a previous formula or to space the feedings further apart. If diarrhea or vomiting occurs, it will be well to revert to homogenized milk alone for a time, before adding the concentrates. This formula is elastic, as additional proteins may be obtained by increasing the amount of amino acids. If less carbohydrate is desired, less dextrimaltose is added. One may decrease the fat in the formula by substituting skim milk and a desired amount of cream for the homogenized milk.

In summary it may be said that forced protein intake is not an easy problem in many patients, but by impressing on them its need, securing their cooperation and by administering products that are well tolerated, we may satisfactorily prepare our patients properly for surgery and maintain them in a fair nutritional state following operation.

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SPONTANEOUS HEMOPNEUMOTHORAX REQUIRING THORACOTOMY

Report of a Case*

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SPONTANEOUS hemopneumothorax is a seldom reported complication of spontaneous pneumothorax. The incidence of this entity is probably more frequent than the literature indicates. There have been about 80 reported cases[2,4] with a mortality rate of approximately 20 per cent.

The advocated treatment has usually been conservative,[5] consisting of sedation, oxygen treatment and diagnostic thoracentesis. Death has usually resulted because of untreated shock due to acute blood loss or mediastinal shift due to unrelieved tension hemopneumothorax. However, with the recent advances in therapy which include antibiotics, adequate blood and electrolyte replacement, positive pressure endotracheal anesthesia and the understanding of cardiorespiratory physiology, this mortality rate should be reduced.[9] The following case is presented as an illustration of the possible serious consequences of this disease, and the relative ease with which it can be handled if the correct principles of modern therapy are applied.

Case Report

T. M., a 19 year old U. S. Marine Corps private, was admitted to the U. S. Naval Hospital, Beaufort, S. C., on Dec. 20, 1950, eight hours following the onset of sudden severe right chest pain and dyspnea. A chest roentgenogram at the U. S. Naval Dispensary revealed evidence of a partial collapse of the right lung and pleural fluid to the level of the tenth rib anteriorly. The past history was relevant in that a chest roentgenogram in September 1949 was said to be normal. A 5- by 7-inch roentgenogram three weeks before admission was not remarkable. The patient had always been underweight and had been subject to frequent upper respiratory infections. There was no family history of any pulmonary disease.

On admission to the hospital physical examination revealed an ambulatory, pale, asthenic young man, 20 pounds under his expected weight. His pulse rate was 112 and respiration rate 32 per minute; blood pressure was 108 mm. Hgb. systolic and 78 diastolic; his temperature was 99.6 F. The trachea was shifted slightly to the left. Breath sounds were absent over the right hemithorax. There was hyperresonance to percussion over the right upper chest

*The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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and dullness to percussion below the sixth rib anteriorly on the right. The left lung was well areated. His heart was not enlarged and did not appear shifted to the left. The remainder of the physical examination was not remarkable except for an undescended left testicle.

An immediate roentgenogram of the chest revealed evidence of hydropneumothorax on the right with complete collapse of the right lung. There was fluid to the level of the fourth rib anteriorly. The mediastinum was in the midline. There were no demonstrable pulmonary parenchymal lesions in the right lung.

Laboratory data: The red blood cells numbered 3,950,000 per cu. mm. The blood hemoglobin content was 12.5 Gm. per 100 cc. The hematocrit was 40 volume per cent. The Kahn test was negative. The urine was not remarkable.

An immediate thoracentesis at the eighth interspace, anterior axillary line yielded 300 cc. of what appeared to be pure blood which clotted on removal. The patient was placed in an oxygen tent and given penicillin, 100,000 units, every three hours. He received sedation as needed.

On the following day, nausea and vomiting were noted. He maintained reasonably stable vital signs with a pulse rate of about 104 per minute, blood pressure of 120 mm. Hgb. systolic and 80 diastolic and respirations of 30 per minute. A repeat blood study showed a drop in the circulating blood volume with an erythrocyte count of 3,400,000 per cu. mm., blood hemoglobin concentration of 10 Gm. per 100 cc. and hematocrit of 33 volume per cent. He was transfused with 500 cc. of whole blood slowly during the day. Late in the day a thoracentesis was done and 700 cc. of blood was removed from the right pleural space.

In the predawn hours of the third hospital day the patient was found to be in shock with a blood pressure of 80 mm. Hgb. systolic and 70 diastolic, a pulse rate of 120 per minute and respirations of 30 per minute. He was semicomatose, pale and cyanotic in the oxygen tent. His red blood cell count was 2,500,000 per cu. mm. with 55 per cent of normal hemoglobin. The trachea had a marked shift to the left. He was transfused with 500 cc. of whole blood and 500 cc. of plasma immediately without appreciable improvement. At this time a surgical consultation was requested. When seen by the surgical staff the patient was semicomatose and cyanotic in the oxygen tent, the cervical veins were distended and the trachea was shifted far to the left. The right chest was dull to percussion and the vital signs were as follows: the blood pressure was 60 mm. Hgb. systolic and 40 diastolic. The pulse rate was 140 per minute. The respirations were 36 per minute. Simultaneously a right thoracentesis and a rapid transfusion were begun. Two thousand cc. of blood were withdrawn from the right pleural space as rapidly as possible with a 50 cc. syringe and a three-way stop clock. As this was being done the trachea shifted back toward the midline, the cyanosis disappeared, the neck veins were no longer distended, the sensorium cleared dramatically and the patient became alert and actually cheerful. Seven hundred and fifty cc. of whole blood were given within a few minutes by transfusion. The blood pressure rose to 110 mm. Hgb. diastolic and 70 systolic. The pulse rate was 95 per minute; the respirations were 24 per minute. A blood count at this time gave the following information: the red blood cell count was 3,400,000 per cu. mm., the hemoglobin was 70 per cent of normal and the hematocrit was 41 volume per cent.

The patient was then carefully followed by serial roentgenograms and blood counts every two hours. Slow transfusion was continued. Within the next

six hours the vital signs remained stable. Color gradually returned as 2,000 cc. of whole blood was given, making a total of 3,500 cc. of whole blood within 24 hours. The patient remained cheerful and cooperative although drowsy. The erythrocyte count remained stable at about 3,500,000 per cu. mm. However, the serial roentgenograms revealed a rather steady rise in the intrathoracic fluid level. Each two hour film revealed that the level had risen one or two interspaces. The patient was then considered to be in stable enough condition to undergo thoracotomy. The steady increase in intrapleural blood after a total of 3,000 cc. had been removed in the past 48 hours indicated a continuing source of bleeding. Just before taking the patient to the operating room he suddenly developed cyanosis, tracheal shift to the left and air hunger. He again lapsed into semiconsciousness. An additional 1,100 cc. of blood was removed by thoracentesis with immediate marked and dramatic improvement. This made a total of 4,100 cc. of blood aspirated prior to operation.

The patient was anesthetized with positive pressure endotracheal gas, oxygen and ether. The right chest was entered through the bed of the fifth rib, a posterolateral approach being used. Approximately 2,500 cc. of clotted and liquid blood was removed from the pleural cavity. At the apex of the parietal pleural there was a recently torn adhesion which was bleeding with a steady flow. This was readily controlled by a figure of eight suture of No. 00 silk.

Inspection of the lung revealed a small torn adhesion at the apex of the right upper lobe without bleeding. No other intrapulmonary lesions could be seen or palpated. The entire right lower lobe was encased in a dense fibrin peel. This was stripped off with ease and the lung then completely reexpanded. The chest was closed in anatomical layers. A water seal catheter drain was placed in the ninth interspace at the posterior axillary line.

The patient's postoperative course was uneventful. The thoracotomy tube was removed on the third postoperative day and the wound healed per primum. The sutures were removed on the seventh postoperative day. The patient received combined chemotherapy of penicillin, streptomycin and aureomycin in appropriate doses for two weeks postoperatively. Beginning on the first postoperative day he received chest exercises and two months postoperatively he had full chest expansion with no functional deformity. Roentgenogram of his chest at this time revealed blunting of the costophrenic angle but the lung was fully expanded and without parenchymal disease.

Pathogenesis

Previous papers have discussed the etiology of this disease.[2,4] Primarily it is considered to be a complication of spontaneous pneumothorax due to tearing of a vascular adhesion between the visceral and parietal pleura. Bleeding may occur from either side of the torn adhesion or bleeding may occur from the wall of a ruptured emphysematous bulla in the pulmonary parenchyna itself. The author's case bled from the parietal side of an apical adhesion. This disease may be, but is usually not, associated with pulmonary tuberculosis.[10]

Clinical Manifestations. The patient usually presents himself

with a history of the sudden onset of chest pain, gradually increasing dyspnea and weakness. He may be cyanotic, dyspneic, anemic and incipient or frank shock may be present, depending upon the rate and amount of blood loss. Abdominal pain has been observed in approximately one-third of the cases and exploratory laparotomy has been done.[7] On physical examination one usually finds a pale, dyspneic, possibly cyanotic individual with splinting of the affected hemithorax. Tracheal shift may be observed. Absence of breath sounds and dullness to percussion of the affected side are to be expected. Abdominal spasm and tenderness with nausea and vomiting may be present. Laboratory studies will reveal that there are varying degrees of anemia. Roentgenogram of the chest will show a hydropneumothorax with varied degrees of pulmonary collapse and a fluid level.

There are two serious immediate effects of continued bleeding into the pleural cavity. First are the consequences of severe blood loss from any source: anemia, depletion of the circulatory volume with resulting peripherovascular collapse and tissue anoxia. These factors alone may be fatal, and they should be energetically treated by blood replacement unit for unit until the measured deficit is made up. When bleeding occurs into the closed pleural space the loss can be accurately measured and thus accurately replaced. It is now generally understood that enough blood should be given to bring the blood pressure and pulse above shock level. Articles of a decade ago feared adequate blood replacement might "blow out" a formed clot and cause further bleeding. Continued tissue anoxia is too high a price to pay for a theory based on no accurate scientific observation. Autotransfusion may be considered in an emergency, but generally speaking it is not wise, for the fluid withdrawn from the chest is not whole blood but has been defibrinated by the action of the heart and lungs;[1,9] it may contain bacteria which have gained entrance through a ruptured bleb, or as is frequently the case, it may be partially hemolyzed.

Bleeding into the pleural space when a spontaneous pneumothorax exists presents problems peculiar to the anatomy and physiology of the region. As the bleeding continues into the closed space, the intrapleural pressure on the affected side rises, causing a further and complete collapse of the lung and eventually a shift of the mediastinum toward the opposite side unless these structures have been fixed by some previous disease. Acute mediastinal shift is a serious consequence in itself causing frequent cardiac arrhythmias, partial obstruction of the venae cavae and reduction of the remaining respiratory reserve—all leading to inadequate oxygenation of the circulating blood and eventual tissue anoxia. It is inter-

esting in the case described that even under close observation the patient very suddenly went into deep shock on two occasions and heroic measures were required to restore equilibrium.

Bleeding into the pleural cavity has late effects due to the organization of the fibrin peel upon the visceral pleura making reexpansion of the collapsed lung difficult if not impossible. The accumulating blood must be aspirated thoroughly and at frequent intervals to avoid these complications, *e.g.,* tension pneumothorax, mediastinal shift and pleural peel. It is the opinion of many authorities that a rapidly and fully expanded lung will eventually give a much greater functional result than a lung that has remained in a state of partial collapse for some weeks and has expanded by the slow absorption of pleural blood and air. While this is occurring, the lung is perforce, partially atelectatic and some degree of pulmonary fibrosis must occur. Those who object to frequent thoracentesis fear empyema. With the proper aseptic technic and the instillation of antibiotics at each thoracentesis this complication should rarely occur.

Repeated thoracenteses and multiple transfusions will in all likelihood suffice as ideal treatment for the majority of instances where spontaneous hemopneumothorax occurs.[6] If the major portion of the defibrinated blood is removed by aspiration, decortication may usually be avoided by vigorous repeated chest exercises initiated by a good physiotherapist. These exercises have been demonstrated to cause an absorption of the pleural peel if begun at an early state and continued for a period of weeks before the chest is "frozen" by organization of the fibrin with dense fibrous tissue.

Occasionally, however, as in bleeding peptic ulcer, a case will be seen in which bleeding continues. If the patient has had continuous adequate blood replacement, thoracotomy can be undertaken and the bleeding stopped without appreciable danger. In this and the single previously reported case of Myers, Johnston and Bradshaw[8] the site of bleeding was on the parietal pleura in the superior sulcus and was readily controlled.

The postoperative course should include closed drainage in the immediate postoperative period, bronchoscopy as indicated for residual atelectasis in the collapsed lung, proper antibiotic therapy and postoperative chest exercises to insure a good cosmetic and functional result.

The case presented above is illustrative of many of the general and specialized points of intrapleural hemorrhage. The patient was anemic and had sufficient hemorrhage to be in shock from blood loss alone. In addition, he had a severe loss of respiratory volume

and a mediastinal shift. By prompt and vigorous thoracentesis and transfusion which restored the circulatory volume and the respiratory reserve, an anoxic, partially comatose man was almost immediately restored to comfort and circulatory balance. There is no reason to believe that mortality from this infrequent complication of spontaneous pneumothorax cannot be reduced to near the zero mark.

Conclusion

1. A case of spontaneous massive hemopneumothorax requiring thoracotomy with recovery is presented.

2. A suggested method of treatment is outlined. It includes thoracenteses, blood replacement and where necessary operative interference.

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THE POTASSIUM PROBLEM IN SURGERY

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POTASSIUM is essential to life. It is the chief cation of the cells, but it has escaped close scrutiny by many physicians, particularly surgeons. Pediatricians have long been cognizant of its important role in babies with diarrhea.[1] There have been many excellent reviews of the entire potassium problem.[2,3,4] The one by Martin and her associates is particularly comprehensive and understandable.[5] Certainly many surgical patients with complicated problems have died of hypopotassemia. Only recently has this fact been recognized and steps taken to prevent such deaths.[6,7,8]

Probably the principal reason for overlooking the value of potassium to the human economy was the erroneous impression that potassium is held within the cell and that very little is able to cross the cell membrane. Actually about two thirds of the intracellular potassium is bound to protein, apparently keeping myosin in solution. The other one third can permeate the cell wall and thus profoundly effect acid-base balance. Conditions producing disintegration of protoplasm such as starvation, wasting diseases, trauma and surgery, result in cellular breakdown and potassium moves out of the cells. Increased adrenal activity associated with the alarm reaction, and ACTH and Cortisone therapy produce cellular liberation of potassium. The process of glycogenolysis carries potassium out of the cell. Alkalosis is the most important condition favoring loss of intracellular potassium. In this condition the plasma sodium is relatively increased. Some of it replaces or drives out, so to speak, the intracellular potassium.

Another reason potassium was neglected for many years was the belief that its administration would result in heart block. This is understandable because it is the concentration of extracellular potassium which produces clinical effects. It is necessary for normal cardiac action and for transmission of nerve impulses in the voluntary and autonomic nervous systems, probably acting at the myoneural junctions. The normal range of concentration in the extracellular fluid is 16 to 22 mg. per 100 cc. (about 4-5.4 mEq). High values do produce heart block and death usually occurs at levels of about 40 mg. per 100 cc. Low values also produces symptoms, and it is this hypopotassemia (hypokalemia) with which we are especially concerned. It is interesting to note that the intracellular concentration of potassium is about 420 mg. per 100 cc. and that 98 per cent

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of the total body potassium, or about 175 Gm., is present in the cells, but it is the 2 per cent within the extracellular fluid or only about 2 Gm., which is significant in the production of symptoms.

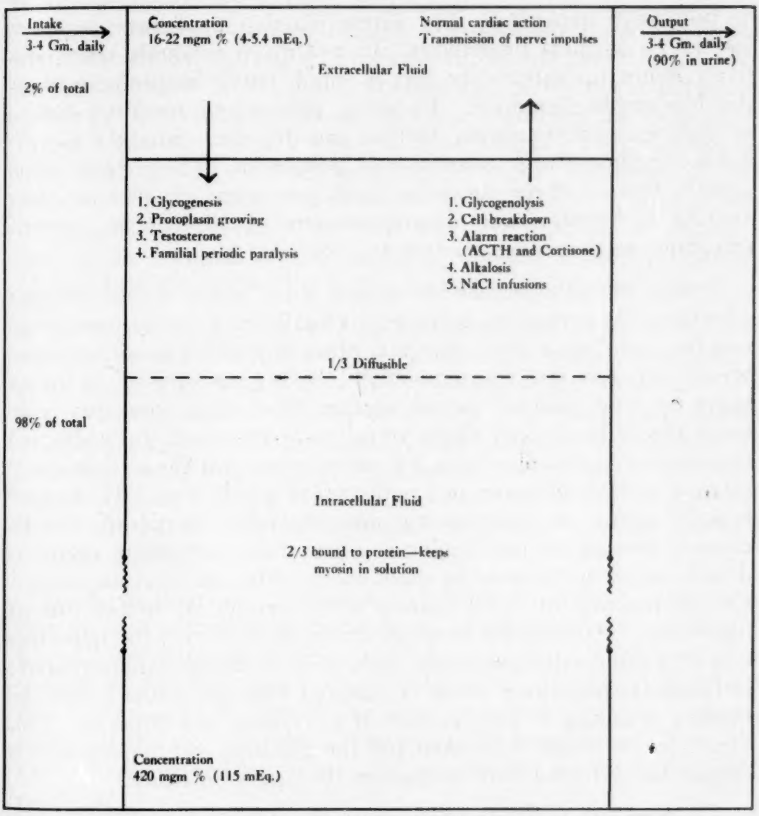

Fig. 1. Potassium Distribution

A normal diet always contains adequate amounts of potassium salts. The average intake is 8 Gm. of potassium chloride daily, supplying about 4 Gm. of potassium. Most of it is absorbed and excreted in the urine, except for small amounts needed for the growth of new cells. The renal tubules ordinarily do not reabsorb potassium as they do sodium, so there is little conservation and there is almost always potassium in the urine. The kidneys can excrete large amounts of potassium supplied to it either by ingestion or by endogenous breakdown of tissue. Figure 1 graphically presents these concepts.

Potassium deficiency is a problem in surgery. Inadequate intake such as in patients with anorexia, carcinoma of the esophagus, some cases of duodenal ulcer, intestinal obstruction, and postoperative restriction of alimentation, is the most frequent clinical cause of potassium depletion. The alarm reaction produces potassium loss in all surgical procedures. In healthy individuals with relatively minor operations the loss is small, but in major operations this loss can be significant. Vomiting, gastric and intestinal suction procedures, gastrointestinal fistulas and diarrhea cause the loss of fluids containing large quantities of potassium. These fluids often contain two or three times as much potassium as an equivalent amount of blood serum. Hypopotassemia itself increases gastric secretions so that more electrolytes are lost.[9]

Several of these mechanisms are at work in the surgical patient who develops potassium deficiency. Usually he has a serious condition necessitating a major surgical procedure which produces tissue breakdown and the alarm reaction. Following surgery he has a short or long gastrointestinal suction tube which removes large quantities of fluid containing electrolytes, particularly chlorides and potassium. More chloride is lost than sodium, so the patient has a relative sodium increase and is therefore in alkalosis. He is now hypochloremic, hypopotassemic and alkalotic. Some of the increased plasma sodium replaces intracellular potassium which is driven out to be excreted by the kidneys. Also, in alkalosis there is altered function of renal tubules which results in further loss of potassium.[10] Now if the surgeon attempts to correct the hypochloremia by using saline solutions, some of the infused sodium replaces intracellular potassium which is excreted with the infused chloride, thereby resulting in further loss of potassium and chloride. This vicious circle cannot be broken and the alkalosis and hypochloremia cannot be corrected until potassium therapy is instituted.

The best way to be alert to potassium deficiency is to be cognizant of these various conditions which produce it. Clinically, such patients are characterized by lethargy, listlessness and extreme weakness with anorexia, nausea, vomiting and ileus being later symptoms. The severity of the symptoms is related to the serum potassium level.

To prove the diagnosis the serum potassium concentration must be measured. Definite symptoms of hypopotassemia occur when the level is down to 12 mg. per 100 cc. At this level electrocardiographic changes occur. These consist of prolonged Q-T interval, depressed S-T segment and a broadened T wave. If chemical or spectroscopic determinations are not available, the serial electro-

cardiographic changes can be interpreted to predict the serum level fairly accurately. Levels below 10 mg. per 100 cc. denote a patient in severe trouble. Below 8 mg. per 100 cc. the patient rarely recovers.

Replacement therapy consists of (1) adequate food, (2) potassium salts by mouth, and (3) potassium chloride parenterally. Any patient who can take a fairly normal diet rarely needs potassium orally or intravenously. Thus, the patient who has not been starving and who has surgery which does not deprive him of food for more than two days will seldom get into trouble. However, if there will be no food intake for more than two days, it is wise to add potassium chloride 1.5 Gm. twice daily to 1000 cc. of whatever intravenous solution is being used. A commercial preparation containing 1.49 Gm. of KCl in a 20 cc. ampule is now available.

If the patient is going to be on gastric suction for more than two days, he should receive 1.5 Gm. of potassium chloride parenterally twice daily. After three days of suction the serum potassium level should be determined. It may be necessary to increase the dosage to 3.0 Gm. parenterally twice daily, depending on the results of the laboratory test. Martin and her associates working on the Potassium Commission at the Los Angeles County General Hospital have demonstrated that larger doses can be given than was formerly thought possible.[11]

If the patient has potassium deficiency when first seen, large quantities of potassium chloride should be given parenterally slowly. Up to 6.0 Gm. can be given in 1000 cc. of glucose or saline solution twice daily, but this amount should seldom be necessary. The patient may have pain at the site of the intravenous infusion and along the course of the vein, but there will seldom be thrombosis. Any solution which is used intravenously can also be given by hypodermoclysis.

When the kidneys are functioning normally there is very little danger of giving too much potassium, because it is so well excreted. However, the solution should be given slowly because of the narrow range of normal extracellular values and the small amount of total serum potassium. Symptoms from too much potassium occur when the serum level rises to about 30 mg. per 100 cc. They are very similar to the symptoms of potassium deficiency. At approximately 40 mg. per 100 cc. cardiac standstill occurs. Hyperpotassemia is associated with impaired renal function, hemoconcentration, rapid tissue disintegration and too rapid parenteral administration of potassium. Consequently, potassium chloride should not be given

or should be given with extreme caution when there is oliguria or anuria.

Case Report

Mrs. L. E., a 47 year old white female, had been on a strict ulcer regimen for many weeks. On July 7, 1948, she had a vagotomy and posterior gastroenterostomy for a duodenal ulcer. During the next eight days suction through a Levine tube removed a total of 8,800 cc. of gastric content from the stomach. During the first ten postoperative days she received 15,000 cc. of normal saline solution intravenously, two-thirds of which was with 5 per cent glucose. She also had 7,000 cc. of 10 per cent glucose in water and four blood transfusions and 2,000 cc. of Amigen. An average of 750 cc. of fluid was being taken orally daily. The urinary output was more than 1500 cc. daily.

Because of increasing abdominal pain and fever, an exploratory laparotomy was done on July 23, 1948, the sixteenth postoperative day. Perforation of the descending colon due to a stercoral ulcer caused by barium in the bowel was found, and a Mikulicz resection of the descending colon was done. Following this surgery the patient did poorly. Levine tube suction was removing an average of 1500 cc. of gastric contents daily for the first eight days. In the first sixteen postoperative days an average of 1800 cc. of normal saline solution was given intravenously daily. Most of this was with 5 per cent glucose. In addition to occasional supportive blood transfusions, a total of 8000 cc. of Amigen was given intravenously. It is calculated that the patient received a total of about 300 Gm. of sodium chloride in the sixteen days. There was a small oral intake of water. The urinary output was more than 1500 cc. daily. Penicillin, streptomycin and intravenous sulfanilamide were being given.

The nurses' notes record hiccoughs on the fourth postoperative day and lethargy on the fifth postoperative day. On the sixth postoperative day the patient had difficulty in swallowing and on the seventh day there was "tingling" of the legs. On the eighth postoperative day there was cramping, abdominal pain and vomiting. From then on the notes are filled with references to drowsiness, nausea and vomiting, abdominal pain, tingling sensations and numbness in the legs. One nurse observed that the tourniquet applied for intravenous administrations occasionally caused carpopedal spasm.

The author saw the patient in consultation on August 9, 1949, the seventeenth day following colostomy. Examination revealed a very lethargic, drowsy, dehydrated woman with marked muscle atonia, fever, an abscess about the colostomy and signs of a left subphrenitis. Chvostek's and Trousseau's signs were negative. Respirations were shallow and the patient was slightly cyanotic. Hypochloremia, alkalosis and hypopotassemia were suspected. The whole blood chlorides were found to be 300 mg. per 100 cc. (laboratory normal 425-500 mg. per 100 cc.), and the blood potassium 7.0 mg. per 100 cc. (normal 16-22 mg. per 100 cc.). The carbon dioxide combining power was 92 volumes per cent. The blood calcium was 10 mg. per 100 cc. During the next ten days the patient received a daily hypodermoclysis of 1000 cc. of a hypotonic salt solution to which was added 6 Gm. of potassium chloride. 1000 cc. of 5 per cent glucose in normal saline was given daily. The blood chlorides and potassium were determined daily and found to slowly rise to normal, while the carbon dioxide combining power slowly fell. The blood sodium was checked one time and found normal. Potassium gluconate grs. 7½ and ammonium chloride grs. 15 were given orally three times daily.

Gastric suction was stopped and the patient was placed on a full diet. The urinary output rose from an average of 1500 cc. daily to nearly 2500 cc. daily.

The patient was observed to definitely improve shortly after the therapy was begun. She became more cheerful and appeared stronger although she was still nervous and restless. She began to eat well and feed herself. Nausea, vomiting and abdominal pain disappeared. The colostomy functioned well and in fact discharged many large, water stools (losing sodium). The paresthesias entirely disappeared. The temperature slowly fell to normal. By August 23, 1948, the patient was walking and gaining strength and vigor, and convalescence was uncomplicated except for the abdominal wall abscess about the colostomy.

Comment

This patient got into severe hypochloremia, hypopotassemia and alkalosis because of lack of food intake, prolonged gastric suction and injudicious use of solutions containing sodium chloride. The potassium level of 7 mg. per cent (1.8 mEq.) is one of the lowest recorded with subsequent recovery of the patient. The quantities of potassium chloride used for therapy were considered large at that time, but we now know even larger quantities could have been used with probably more rapid clinical and laboratory improvement.[11]

Summary

Potassium deficiency is a serious problem in some surgical patients. It is usually brought about by a combination of factors, the chief of which are: (1) no food intake, (2) gastrointestinal suction procedures, (3) alkalosis and (4) injudicious use of parenteral saline solutions. It can be successfully treated by prompt therapy with large amounts of potassium chloride parenterally.

A case illustrating these principles is presented.

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EDUCATIONAL STEPPING-STONES TO SURGERY

The space of a single lifetime has seen the transformation of the training of a medical specialist from a two-year lecture course to a 10- to 14-year program of intensive participating study. The wedding of the University and the Hospital has been crucial to this growth, and has established certain natural stages of training. Although these stages are rather uniform in this country, there appears to be no general agreement as to either the ultimate objective of surgical training, or the immediate and cumulative aims of the various steps.

The fundamental ultimate aim of the training of surgeons can be clearly stated. It is to improve the quality of surgical care given the people of this country by improving the quality of the practitioner of the art. In the long run, surgery should be done by surgeons. The objectives of each stage of training must progress in steplike fashion toward this end.

A. *Premedical Preparation.* The objective of the college experience should be to expose the student to a broad humanistic education designed to enlarge his intellectual and moral horizons, to im-

plant the drive toward continuing self education, and to broaden his understanding of the activities of mankind.

B. *Medical School Preparation.* The objective of the medical school course in surgery should be to familiarize the student with the current scope and future potentialities of surgery in the treatment of disease. It is essential that he grasp the fact that the basis of surgery is in the biological sciences. To this end, anatomy, pathology, and physiology, as they relate to the care of the patient, must be heavily emphasized. Of surgical technic, only the principles should be taught. Anesthesia, asepsis, hemostasis, and gentleness with living tissue—these are fundamental to all manipulative procedures in medicine from venipuncture to thoracotomy. On the other hand, an acute awareness of the pitfalls, difficulties, complications, and dangers of operative therapy must be instilled.

At the conclusion of his medical school career, therefore, no student should be considered capable of even the most simple surgical procedures.

C. *The Internship.* The objective of the internship is to develop the student as a physician. To this end, his clinical experience in the diagnosis and management of people with real or imagined disease must be enriched, and his sense of unending obligation to the needs of his patient must be firmly ingrained. During his short tour on the surgical service, this aim is accomplished by emphasizing the application of the scientific bases of surgery in careful pre- and postoperative care of the patient. Little technical training is desirable or possible during this short period.

At the conclusion of his internship, the student has begun to mature as a physician, but is still incapable of any surgical procedures unsupervised.

D. *The Residency* (graduate training in surgery). The objective of the residency is to produce a practicing physician capable of responsible operative intervention in the treatment of the sick patient, aware of his obligations toward the well-springs of his profession—teaching and research. To this end, a broad guided technical experience leads to progressive operative responsibility, always with insistence on the strict self discipline demanded by the needs of the patient and the moral integrity to do, with courage, only those procedures honestly felt to be for the benefit of the patient. Critical analysis of the reasons for action helps to imbue a continuing interest in the sources and methods of dissemination of professional knowledge, and a sense of obligation to promote the standards of his specialty.

At the conclusion of his residency, the student is capable of skillful responsible operative treatment of a wide variety of human ailments, and is interested in bringing only the best to his patient.

Thus, it is argued, the proper training for either the practicing surgeon or the academic teacher is essentially the same. In the past, the Halsted or University type residency has been geared to produce teachers, the "clinical" residencies to produce "practical" surgeons. The need in America is for a wide distribution of men whose training represents a combination of both, fitting them to assume the responsibilities of patient care and hospital leadership in the community hospitals throughout the country, practicing often in the organizational setting of group medicine. Through the efforts of such men, widely scattered throughout the country, the standards of surgical practice could best be elevated. This ultimate objective of surgical training must be sought in action in the arena beyond the educational pathway. Those responsible must see that the stepping-stones lead to the gateway.

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BOOK REVIEWS

The Editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

THE MANAGEMENT OF FRACTURES, DISLOCATIONS, AND SPRAINS. By JOHN ALBERT KEY, B.S., M.D., Clinical Professor of Orthopedic Surgery, Washington University School of Medicine; Associate Surgeon, Barnes, Children's and Jewish Hospitals, and H. EARLE CONWELL, M.D., F.A.C.S., Associate Professor of Orthopedic Surgery, University of Alabama School of Medicine, Birmingham, Ala. St. Louis, C. V. Mosby Company, 1951. Cloth binding. Illus. 1232 pages. $16.00.

Perhaps no two men are better qualified to write on the subject of trauma than the authors of this book. Both have had considerable experience and have contributed much to our present knowledge of fractures.

It is a textbook which improves with each new edition. The chapter on compound fractures and war wounds is a valuable one and is nicely illustrated. All of the basic principles are duly stressed. Intramedullary fixation of the long bones, notably the femur and tibia, is discussed and illustrated at length.

This edition is written in the same clear and concise manner as the previous editions and the author states that the changes have been so extensive that the entire book has been reset. Illustrations have been improved remarkably. This book will remain as one of the best of all textbooks on fractures and, as such, should be enthusiastically read by all interested in this subject.

WOODROW W. LOVELL, M D.

CONGENITAL DISLOCATION OF THE HIP. By JULIUS HASS, M.D., Consulting Orthopedic Surgeon at Montefiore Hospital, New York City; formerly Professor and Chief of the Orthopedic Clinic of Vienna, Austria. Springfield, Ill., Charles C Thomas, 1951. Cloth binding. 405 pages. Illus. $12.50.

The author of this book has personally observed more than 2,000 cases of congenital dislocation of the hip. This, in itself, is amazing. Few, if any, in a lifetime will see this number of congenital dislocated hips. Heretofore, there has been considerable confusion as to the difference between prenatal and postnatal cases. Dr. Hass very clearly differentiates between the two.

A very interesting chapter is devoted to etiology. One of the most valuable chapters is concerned with pathology. In no other manuscript on this condition is the pathology so beautifully explained and illustrated.

The final chapters are concerned with treatment and the author discusses closed reduction, open reduction, and reconstructive shelf procedures.

In summary, this book is easily readable, of good print, clearly illustrated and contains a lengthy bibliography. It should and will be accepted as a classic on this interesting and challenging entity.

WOODROW W. LOVELL, M.D.

Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

EARLY CARE OF THE SERIOUSLY WOUNDED MAN. By HENRY K. BEECHER, M.D., Chief, Department of Anesthesia the Massachusetts General Hospital, Boston, Mass. Edited by Michael E. De Bakey, M.D., Professor of Surgery, Chairman of the Department of Surgery, Baylor University College of Medicine, Houston, Texas, and R. Glen Spurling, M.D., Clinical Professor of Surgery, University of Louisville, Louisville, Ky. Springfield, Charles C Thomas, 1952, 32 pages, $.75.

UROLOGICAL PATHOLOGY. By PETER A. HERBUT, M.D., Professor of Pathology, Jefferson Medical College and Director of Clinical Laboratories, Jefferson Medical College Hospital, Philadelphia, Pa. Volume I, 527 illustrations, 693 pages; Volume II, 527 illustrations, Philadelphia, Lea & Febiger, 1952. $24.

PRESCRIPTION FOR MEDICAL WRITING. By EDWIN P. JORDAN, M.D., and WILLARD C. SHEPARD, M.D., Philadelphia, W. B. Saunders Company, 1952. $2 50.

THE CLINICAL USE OF FLUID AND ELECTROLYTE. By JOHN H. BLAND, M.D., Assistant Professor of Medicine, University of Vermont College of Medicine. Illustrated, Philadelphia, W. B. Saunders Company, 1952. $6.50.

A TEXTBOOK OF ORTHOPEDICS with a section on Neurology in Orthopedics. By M. BECKETT HOWORTH, M.D., Clinical Professor of Orthopedic Surgery, New York University Postgraduate Medical School. In association with: Fritz J. Cramer, M.D., Donovan J. McCune, M.D., A. Wilbur Duryee, M.D., J. William Littler, M.D., Walter A. Thompson, M.D. Philadelphia, W. B. Saunders Company, 1952. $16.

ABSTRACTS FROM CURRENT LITERATURE

HIATAL HERNIA. A CAUSE OF PERSISTENT GASTROINTESTINAL DISTURBANCES IN PREGNANCY. W. Robert Penman. *Western Journal of Surgery, Obstetrics and Gynecology 59*:622-625 (Dec.) 1951.

Following a review of the literature on the subject, Penman describes 3 cases of hiatal hernia complicating pregnancy which were diagnosed in the prenatal period. In a discussion of the management of this complication, the author emphasizes the employment of bland diet with frequent small feedings, hospitalization early in labor and consummation of delivery as soon as the cervix is dilated. In event vomiting develops, the patient should be hospitalized and in the absence of radiological evidence of obstruction, the patient should still be managed conservatively with intubation and suction plus intravenous fluids. In the case of obstruction, intubation with suction, nasal oxygen and parenteral nutrition should be employed for a reasonable length of time. If there is no response within a reasonable period, surgery is mandatory.

Strangulated hiatal hernia in pregnancy carries a very high mortality rate. Early surgical intervention with resection of gangrenous or nonviable bowel will greatly reduce this.

R. H. S.

SURVIVAL FIVE YEARS AFTER RADICAL PANCREATODUODENECTOMY FOR CARCINOMA OF THE HEAD OF THE PANCREAS. Edward M. Miller and O. Theron Clagett. *Annals of Surgery 134*:1013-1017 (Dec.) 1951.

Herein is reported a case of carcinoma of the head of the pancreas treated by radical pancreatoduodenectomy followed at the Mayo Clinic for a period of five years, the patient being well, working and free of evidence of disease at the time of this report. This is the first five year survival reported for this condition though several recent reports of five year survival after pancreatoduodenectomy performed for carcinoma of the ampulla of Vater are cited.

Prior to July, 1945, 11 patients with carcinoma of the head of the pancreas were treated at the Mayo Clinic by pancreatoduodenectomy. Of these, 2 died in the immediate postoperative period, and 6 died 12 months or less following operation. One died 22 months after operation and another succumbed 44 months after surgery. On the basis of the 1 patient surviving at present, the five year survival rate is computed at 9 per cent.

The authors conclude that radical pancreatoduodenectomy holds promise in the treatment of cancer of the head of the pancreas. They question whether total pancreatectomy, in view of its more crippling after effects, is sufficiently more radical than partial pancreatectomy to justify complete resection of the organ.

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